# Self-Assessment in Multiprofessional Critical Care

## A Comprehensive Review

6th Edition

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# Self-Assessment in Multiprofessional Critical Care A Comprehensive Review 6th Edition

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Managing Editor: Christine Mentink Editorial Assistant: Kimberly A. Hoppe

Printed in the United States of America First Printing, August 2007

Society of Critical Care Medicine

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International Standard Book Number: 978-0-936145-28-0

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# SECTION 1: Airway Management/ Anesthesiology

# SECTION 1: AIRWAY MANAGEMENT/ ANESTHESIOLOGY

**Instructions:** For each question, select the most correct answer.

A 65-year-old male has been admitted to the ICU following a Whipple procedure. An epidural
catheter was placed preoperatively for postoperative analgesia. Upon admission, his drug regimen
included a low-molecular-weight heparin administered every 12 hours for the prophylaxis of deep
venous thrombosis. The epidural was inadequate for his pain control, and he was placed on patient
controlled analgesia.

How long after the last dose of low-molecular-weight heparin is given is it safe to remove the epidural catheter?

- A. Immediately
- B. 4 hours after the last dose
- C. 12 hours after the last dose
- D. 24 hours after the last dose
- E. 36 hours after the last dose
- A 73-year-old female is 1 postoperative day after a left upper lobectomy. She has been receiving continuous epidural analgesia with 0.1% bupivacaine with 10 μg fentanyl/mL at 5 mL/h. In the morning, she is somnolent but will awaken with stimulation. The morning arterial blood gas was 7.28/54/100 on 2 L/min nasal cannula. Her vital signs are BP 136/84 mm Hg, HR 62/min, RR 8/min.

Which one of the following represents the most appropriate intervention?

- A. Reduce the epidural infusion rate to 2 mL/h
- B. Change the epidural infusion to 0.1% bupivacaine without a narcotic
- C. Administer 80 µg naloxone
- D. Remove the epidural catheter and substitute patient controlled analgesia
- E. Supply supplemental oxygen with a face mask

3. Following a high speed motor vehicle collision, a 25-year-old male was intubated in the field with a nasotracheal tube. Injuries included a C3 vertebral body fracture that was stabilized with a halo vest. On ICU day 3, he developed a temperature of 39.6°C (97.8°F). When a CT scan of the head revealed bilateral opacification of the maxillary sinuses, the decision was made to remove the nasotracheal tube and intubate the trachea via the orotracheal route.

Which one of the following would be the best method to accomplish this?

- A. Remove the nasotracheal tube and use direct laryngoscopy to place an endotracheal tube through the mouth
- B. Exchange the endotracheal tube with a reintubation stylet
- C. Remove the nasotracheal tube, ventilate the patient with an intubating laryngeal mask airway (LMA), followed by intubation through the LMA
- D. Pass a fiberoptic bronchoscope into the trachea through the mouth, remove the nasotracheal tube, and intubate over the bronchoscope
- E. Leave the nasotracheal tube in place until a tracheostomy can be performed
- 4. Following unsuccessful treatment with inhaled bronchodilators and systemic steroids, an 83-year-old female with chronic obstructive pulmonary disease, type 2 diabetes mellitus, and a cardiomyopathy (left ventricular ejection fraction 23%) required endotracheal intubation for respiratory distress that was secondary to severe bronchospasm. Intubation was facilitated with an IV dose of 20 mg etomidate. After a 6-hour period of hemodynamic stability, she became progressively hypotensive and required pressor support. Which one of the following represents the most likely etiology of her hypotension?
  - A. Sepsis
  - B. Hypovolemia
  - C. Adrenal insufficiency
  - D. Exacerbation of her cardiomyopathy
  - E. Decreased venous return secondary to mechanical ventilation

5. Following an uneventful thoracoabdominal aneurysm repair, a 70-year-old female with chronic obstructive pulmonary disease, chronic renal insufficiency, and peripheral vascular disease remains intubated and mechanically ventilated. She is sedated with an infusion of propofol at 40 μg/kg/min. Hydromorphone (1 mg every 6 hours) is given for analgesia. Her BP is 180/90 mm Hg with a HR of 105-115/min. After the propofol infusion is increased to 80 μg/kg/min, her BP decreases to 150/84 mm Hg, while the HR remains 102-114/min.

Which intervention to control the hemodynamics should be included?

- A. Increasing the propofol infusion to provide analgesia
- B. Increasing the hydromorphone dose and frequency for analgesia
- C. Adding lorazepam 1 mg q 8 hour for additional sedation
- D. Adding haloperidol 2 mg IV for additional sedation
- E. Administration of hydralazine for blood pressure control
- 6. After a gun shot wound to the left chest, a 19-year-old male is admitted to the ICU following an exploratory thoracotomy and laparotomy. He remains intubated and mechanically ventilated with an Fio<sub>2</sub> of 0.4, pressure control 18 cm H<sub>2</sub>O, rate 12/min, and positive end expiratory pressure 5 cm H<sub>2</sub>O. The total respiratory rate is 14/min. There is a small air leak present in the water seal of the chest tube collection system. This air leak progressively increases to the point that the patient cannot be ventilated with adequate tidal volumes.

What is the best immediate strategy to maintain adequate ventilation?

- A. Change to Fio<sub>2</sub> 1.0, synchronous intermittent mandatory ventilation 12, Vo 500, rate 12/min, positive end expiratory pressure 5 cm H<sub>2</sub>O
- B. Advance the endotracheal tube into the right mainstem bronchus
- C. Replace the endotracheal tube with a double lumen endotracheal tube in order to isolate the left lung
- D. Change to Fio<sub>2</sub> 1.0, pressure support 18, positive end expiratory pressure 5 cm H<sub>2</sub>O
- E. Administer a neuromuscular blocker to facilitate mechanical ventilation

7. A 64-year-old male is status post an Ivor-Lewis esophagectomy for adenocarcinoma of the esophagus. This procedure consists of an exploratory laparotomy to mobilize the stomach and a right thoracotomy to excise the lesion and create a gastroesophageal anastomosis. For optimal surgical exposure, one-lung ventilation is needed intraoperatively to collapse the right lung. Because he was a difficult endotracheal intubation and had an unexpectedly prolonged procedure, he remained intubated with a left double lumen endotracheal tube. The tube was partially withdrawn so that the distal tip is now in the trachea. During the first postoperative evening, he has frequent episodes of coughing. The morning chest radiograph reveals right upper lobe atelectasis.

What is the most likely etiology?

- A. Periodic movement of the left endotracheal tube into the right mainstem bronchus
- B. Occlusion of the trachea
- C. Mucous plugging

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- D. Insufficient positive end expiratory pressure
- E. Insufficient chest tube suction
- 8. A 60-year-old male with a history of hypertension, chronic renal insufficiency, and type 2 diabetes mellitus is involved in a high-speed motor vehicle collision. Injuries included a closed head injury and a liver laceration that was emergently repaired. Because of significant edema of the intestines that is secondary to a massive transfusion, the surgical team elects to delay closure of the abdomen and requests paralysis of the patient with a holiday for daily neurologic examinations.

What is the neuromuscular blocker best suited for this patient?

- A. Pancuronium
- B. Cisatracurium
- C. Vecuronium
- D. Pipecuronium
- E. Mivacurium

- 9. Which one of the following is not a relative contraindication for percutaneous tracheostomy?
  - A. Aggressive mechanical ventilator support (positive end expiratory pressure of 10 cm H<sub>2</sub>O or greater) or high oxygen concentration (60% or greater)
  - B. Unstable cervical spine
  - C. Goiter
  - D. Mediastinal irradiation
  - E. Upper airway obstruction
- 10. Which one of the following is a typical characteristic of etomidate when used as an induction agent for endotracheal intubation?
  - A. Decrease in BP
  - B. Histamine release
  - C. Apnea
  - D. Inhibition of adrenal steroidogenesis
- 11. To facilitate an endotracheal intubation, propofol, and succinylcholine were given to a 56-year-old male in respiratory failure. There were preintubation findings that would predict a difficult airway. The first and second intubation attempts by the ICU resident were unsuccessful. Oxyhemoglobin saturation is maintained normal with bag-mask ventilation.

What is the next most appropriate step?

- A. Bag-mask ventilation followed by a third intubation attempt by the resident
- B. Bag-mask ventilation followed by placement of a laryngeal mask airway
- C. Laryngeal mask airway insertion without mask ventilation
- D. Bag-mask ventilation and an intubation attempt by a more experienced physician
- E. Bag-mask ventilation until the patient recovers from the propofol and succinylcholine, followed by fiberoptic-guide endotracheal intubation

- 12. A thoracoabdominal esophagectomy is planned for a 46-year-old male with an otherwise unremarkable past medical history. Unfortunately, the laparotomy revealed diffuse metastatic disease and the procedure was aborted. He is brought to the ICU because he was still chemically paralyzed. Prior to extubation, which of the following would confirm return of muscle strength?
  - A. A return of the train-of-four to 4 equal, strong, twitches
  - B. Inspiratory force of -20 cm H<sub>2</sub>O
  - C. 600 mL tidal volume on pressure support ventilation at 16 cm H<sub>2</sub>O
  - D. Ability to follow commands
  - E. RR greater than 18/min
- 13. Two hours after chemical paralysis has begun to reduce the abdominal wall pressure of a patient with abdominal compartment syndrome, the bispectral index (BIS) is 90. Which one of the following is the most appropriate intervention?
  - A. Increase analgesia with hydromorphone
  - B. Increase sedation

- C. Increase the level of neuromuscular blockade until the BIS is lowered to 40
- D. Reverse the neuromuscular blockade and transition the patient to pressure support ventilation
- E. No intervention needed, because a BIS of 90 is normal
- 14. Twenty-four hours after a laparoscopic removal of an ectopic pregnancy, a 28-year-old female is admitted to the ICU. Her primary postoperative problem is nausea, which is treated with promethazine. However, over several hours, she becomes febrile, with a maximum temperature of 39°C (102.2°F) and is increasingly agitated. Laboratory studies indicate a white blood cell count of 35,000/μL.

When considering a diagnosis of neuroleptic malignant syndrome, which one of the following is correct?

- A. It has the same cellular basis as malignant hyperthermia
- B. Neuroleptic malignant syndrome occurs only in patients receiving psychotropic medications
- C. It is often self-limiting if the triggering agent is stopped and requires no further therapy
- D. The leukocyte count is not elevated
- E. The diagnosis is often delayed and easily missed

- 15. Which of the following is a property of dexmedetomidine?
  - A. Respiratory depression
  - B. Antagonism at  $\alpha_1$  receptors
  - C. Agonist action at α, receptors
  - D. Prolongation of the QTc with cumulative dosing
  - E. Ganglionic blockade in high doses
- 16. A 62-year-old male with myasthenia gravis is admitted to the ICU after a tree limb fell on his left lower extremity. He has multiple facial lacerations and a possible open globe injury from the tree's branches. Pertinent laboratory studies show a +4 heme in urine and a creatinine of 3.8 mg/dL (335.92 μmol/L). Prior to transport to the operating room for urgent ocular and leg explorations, he becomes hemodynamically unstable and requires endotracheal intubation.

Which one of the following represents a contraindication to the use of succinylcholine?

- A. Renal insufficiency
- B. Open globe injury
- C. Crush injury
- D. Myasthenia gravis
- E. Facial lacerations
- 17. A 56-year-old female is admitted to the ICU for observation after a syncopal episode in a shopping mall. Her only injury was a nasal fracture. Her past medical history and allergies to medications are unknown, but she has tremors and choreiform movements that are compatible with Parkinson's disease.

Which one of the following analgesics should not be used for pain control in this patient?

- A. Morphine
- B. Hydromorphone
- C. Meperidine
- D. Acetaminophen
- E. Ketorolac

18. Following a motor vehicle collision, a 20-year-old male is admitted to the ICU. Injuries include a fracture of the left humerus and a possible closed head injury (Glasgow coma scale score 11). He is in a cervical collar. Within the first hour, his neurologic status deteriorates to Glasgow coma scale score 7, and he requires endotracheal intubation for airway protection.

In order to accomplish this intubation, which of the following sequences provide the optimum conditions?

- A. No sedation, cervical collar in place, fiberoptic intubation
- B. No sedation, cervical collar in place, direct laryngoscopy
- Rapid sequence induction with propofol/succinylcholine, in-line manual stabilization, cervical collar off, direct laryngoscopy
- D. Rapid sequence induction with etomidate/vecuronium, in-line manual stabilization, cervical collar in place, direct laryngoscopy
- E. Propofol, in-line manual stabilization, fiberoptic intubation
- 19. Following a modified radical neck dissection, a 72-year-old male is admitted to the ICU for routine progressive care. Pertinent past medical history includes hypertension treated with lisinopril. Shortly after admission, the nurse notices that his tongue appears to be larger than upon admission. When you arrive, the nurse informs you that the tongue has now doubled in size since you were called to evaluate him. The patient has become increasingly anxious.

What is the most appropriate treatment at this time?

- A. Nebulized epinephrine
- B. Diphenydramine 50 mg IV and dexamethsone 12 mg IV
- C. Observation

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- D. Immediate intubation by an anesthesiologist with a surgeon present
- E. Elevation of the head of the bed 30°

- 20. Which one of the following is the best drug combination for a sedated and paralyzed patient with end stage renal disease?
  - A. Morphine, vecuronium
  - B. Morphine, cisatracurium
  - C. Hydromorphone, vecuronium
  - D. Hydromorphone, cisatracurium
  - E. Hydromorphone, pancuronium
- 21. A 25-year-old male with a previous history of a colectomy for ulcerative colitis arrives in the ICU following an uneventful emergent exploratory laparotomy analysis of adhesions for a small bowel obstruction. He remained intubated and was placed on Fio<sub>2</sub> 0.4, pressure control 10 cm H<sub>2</sub>O, rate 12/min, positive end expiratory pressure 5 cm H<sub>2</sub>O. Thirty minutes after arrival, he has a sudden onset of tachypnea (40-50 breaths per minute), followed by fever and ventricular ectopy on the ECG monitor. Capnography reveals a rise in the end-tidal CO<sub>2</sub> from 42 mm Hg on admission to 80 mm Hg.

Which one of the following is the most appropriate treatment at this time?

- A. A neuromuscular blocker to control his ventilation
- B. An opiate as needed to control pain
- C. An antipyretic to reduce fever
- D. Dantrolene
- E. Sedation with an infusion of propofol

22. A 57-year-old male is admitted to the ICU for an acute myocardial infarction. Several hours after admission, his cardiac rhythm changed from a sinus tachycardia to a ventricular fibrillation. Immediate defibrillation did not change the rhythm. He was orally intubated, and chest compressions were started. The patient was ventilated by an bag-mask device with 100% oxygen at a rate of 18/min.

On examination, no rush of air was heard in the stomach with bad compression, breath sounds were equal bilaterally, and there was symmetrical chest wall movement. Using a colorimetric capnometer, a low level of end-tidal CO<sub>2</sub> is detected immediately after intubation. End-tidal CO<sub>2</sub> remained detectable at a level of approximately 0.5% during the next 5 minutes of resuscitation.

What is causing the persistent low level of end-tidal CO, at 0.5%?

- A. Low cardiac output
- B. Esophageal intubation
- C. Hypoventilation
- D. High cardiac output
- E. Inadequate bag-mask device O, flow

### SECTION 1: AIRWAY MANAGEMENT/ ANESTHESIALOGY

ANSWERS:

1-C; 2-B; 3-D; 4-C; 5-B; 6-B; 7-A; 8-B; 9-E; 10-D; 11-D; 12-A; 13-B; 14-E; 15-C; 16-C; 17-C; 18-C; 19-D; 20-D; 21-D; 22-A

RATIONALE (1) Answer: C

Patients who have received low-molecular-weight heparin (LMWH) are assumed to have altered coagulation. Twelve hours following injection, 50% of the peak anti-Xa activity will be present. In patients treated with LMWH, the risk of causing an epidural hematoma during either the placement or removal of an epidural catheter is high. Therefore, for patients administered LMWH doses every 12 hours, the catheter can be removed 10-12 hours after the anticoagulant is given. The LMWH can be restarted 2 hours after catheter removal.

#### **REFERENCES (1)**

Faccenda KA, Finucane BT. Epidural block: technical aspects and complications. *Curr Opin Anaesthesiol* 2002;15:519-523.

Gogarten W. The influence of new antithrombotic drugs on regional anesthesia. *Curr Opin Anaesthesiol* 2006; 19:545-550.

Horlocker TT, Wedel DJ. Anticoagulation and neuraxial block: historical perspective, anesthetic implications, and risk management. *Reg Anesth Pain Manag* 1998; 23(suppl 2):129-134.

Horlocker TT, Heit JA. Low-molecular-weight heparin: biochemistry, pharmacology, perioperative prophylactic regimens, and guidelines for regional anesthetic management. *Anesth Analg* 1997; 85:874-885.

Llau JV, De Andres J, Gomar C, et al. Anticlotting drugs and regional anaesthetic and analgesic techniques: Comparative update of the safety recommendations. *Eur J Anaesthesiol* 2007;24:387-398.

Tam NL, Pac-Soo C, Pretorius PM. Epidural haematoma after a combined spinal-epidural anaesthetic in a patient treated with clopidogrel and dalteparin. *Br J Anaesth* 2006;96:262-265.

RATIONALE (2) Answer: B

Low respiratory rate and hypercarbia indicate that this patient has respiratory depression. When opiates are given as part of an epidural analgesic regimen, respiratory depression is a common result, caused by the systemic absorption of opitates from the epidural space. Epidural fentanyl infusions are equivalent to IV administration. Because this patient was stable and not in acute respiratory distress, changing the epidural infusion to 0.1% bupivacaine without an opiate should improve this patient's respiratory status.

Although lowering the rate would limit the total opiate given, it would also limit the amount of local anesthetic and might not provide adequate analgesia.

#### **REFERENCES (2)**

- Logas KA, Ready LB, Downey M. Epidural and intravenous fentanyl infusions are clinically equivalent after knee surgery. *Anesth Analg* 1990;70:72-75.
- Sandler AN, Stringer D, Panos L, Badner N, et al. A randomized, double-blind comparison of lumbar epidural and intravenous fentanyl infusions for postthoracotomy pain relief. Analgesic, pharmacokinetics, and respiratory effects. *Anesthesiology* 1992;77:626-634.
- Wheatley R, Somerville I, Sapsford D, Jones J. Postoperative hypoxemia: Comparison of extradural, I.M., and patient-controlled analgesia. *Br J Anaesth* 1990;64:267-275.

RATIONALE (3)

Answer: D

Halo vests that use rigid rods to connect a ring encircling the head to a chest plate are often used to immobilize cervical spines with unstable vertebral fractures. If intubation or re-intubation of a patient is needed, the fracture and this immobilization device can create a difficult airway. Emergent or semi-emergent intubation of patients in halos is associated with significant morbidity and mortality. Direct laryngoscopy is not possible. A re-intubation stylet would not be effective, because the nasal tube is being exchanged for an oral one. The laryngeal mask airway in this scenario would be a rescue technique in the event of an unanticipated extubation. The safest approach is to remove the nasotracheal tube, then use a fiberoptic bronchoscope to visualize the trachea and permit intubation.

#### **REFERENCES (3)**

- Manoach S, Paladino L. Manual in-line stabilization for acute airway management of suspected cervical spine injury: historical review and current questions. *Ann Emerg Med* 2007; (In print).
- O'Connor MF, Ovassapian A. Management of the airway and tracheal intubation. In Murray MJ, Coursin DB, Pearl RG, Prough DS, eds. *Critical Care Medicine*. Philadelphia: Lippincott; 2002:122-136.
- Rosen P, Sloane C, Ban KM, et al. Difficult airway management. *Intern Emerg Med* 2006;1:139-147. Sims CA, Berger DL. Airway risk in hospitalized trauma patients with cervical injuries requiring halo fixation. *Ann Surg.* 2002;235:280-284.
- Stone DJ, Bogdonoff DL. Airway considerations in the management of patients requiring long-term endotracheal intubation. *Anesth Analg.* 1992;74:276-287.
- Walz JM, Zayaruzny M, Heard SO. Airway management in critical illness. Chest 2007;131:608-620.

RATIONALE (4)

Answer: C

Etomidate is a carboxylated imidazole that is used as an anesthetic induction agent. It does not cause hypotension or reduction of cerebral blood flow and does not release histamine. Because of this cardiovascular stability, etomidate is often used to facilitate endotracheal intubation of patients with severe cardiac disease. Side effects of etomidate include pain on injection, nausea, and myoclonus. Etomidate will suppress steroid synthesis in the adrenal cortex. Although adrenal suppression has been seen primarily following infusions (a method no longer in use), it may occur after a single dose, especially in a patient who may have been previously medicated with steroids.

#### **REFERENCES (4)**

Harrison NL, Sear JW. Intravenous anesthetics. Barbiturates, etomidate, propofol, ketamine, and steroids. In Evers AS, Maze M, eds. *Anesthetic Pharmacology: Physiologic Principles and Clinical Practice*. Philadelphia, PA: Churchill Livingstone; 2004:395-416.

Ostwald P, Doenicke AW. Etomidate revisited. Curr Opin Anaesthesiol. 1998;11:391-398.

Petrie J, Glass P. Intravenous anesthetics. Curr Opin Anaesthesiol. 2001;14:393-397.

Young CC, Prielipp RC. Sedation, analgesia, and neuromuscular blocking drugs. In Murray MJ, Coursin DB, Pearl RG, Prough DS, eds. *Critical Care Medicine*. Philadelphia, PA: Lippincott; 2002:147-167.

RATIONALE (5)

Answer: B

Propofol, benzodiazepines, and neuroleptics have no analgesic properties. This patient is receiving narcotic analgesia on a 6-hour schedule. Inadequate pain control in this sedated patient may be one reason for her increased blood pressure and tachycardia. Increasing the dose and frequency of the hydromorphone would provide better analgesia and lead to more stable hemodynamics. The doses employed for this patient are comparable to those used for sedation in the operating room and will usually be sufficient to sedate an elderly patient with comorbidities.

#### **REFERENCES (5)**

Chang AK, Bijur PE, Meyer RH, Kenny MK, Solorzano C, Gallagher EJ. Safety and efficacy of hydromorphone as an analgesic alternative to morphine in acute pain: a randomized clinical trial. *Ann Emerg Med* 2006;48:164-172.

Harrison NL, Sear JW: Intravenous anesthetics. Barbiturates, etomidate, propofol, ketamine, and steroids. In Evers AS, Maze M, eds. *Anesthetic Pharmacology: Physiologic Principles and Clinical Practice*. Philadelphia, PA: Churchill Livingstone; 2004:395-416.

Petrie J, Glass P. Intravenous anesthetics. Curr Opin Anaesthesiol 2001;14:393-397.

Steiner LA, Johnston AJ, Chatfield DA, et al. The effects of large-dose propofol on cerebrovascular pressure autoregulation in head-injured patients. *Anesth Analag* 2003;97:572-576.

RATIONALE (6)

Answer: B

Although significant CO<sub>2</sub> elimination occurs in air passing through a bronchopleural fistula, the patient may be suffering from a life-threatening lack of adequate ventilation. In order to effectively ventilate this patient, the parenchymal or bronchial air leak causing the loss of tidal volume in the effective lung must be bypassed. This can be accomplished by isolating the ventilation to the left lung by selective intubation of the right mainstem bronchus, using a double lumen endotracheal tube of the type often employed for thoracic surgical procedures. However, this requires expertise, is time consuming, and will necessitate removing the existing single lumen endotracheal tube. This process may not be tolerated by a patient in critical condition.

Because the bifurcation of the trachea into the right mainstem bronchus has less of an acute angle than that of the left, it is easy for an endotracheal tube to be advanced into the right mainstem bronchus, either blindly or using fiberoptics. This will provide effective one-lung ventilation until the source of the air in the contralateral thorax can be corrected. Ventilation to the right upper lobe is often sacrificed with a right endobronchial intubation because the anastomosis of the right upper lobe bronchus to the right mainstem bronchus is just distal to the carina.

In a similar fashion, for a right-sided lesion, isolation of the lung can be accomplished using selective ventilation with a double lumen tube (clamping the tracheal lumen of a left-sided double lumen tube), or with endobronchial intubation of the left mainstem bronchus using the fiberoptic guidance of a standard endotracheal tube.

#### REFERENCES (6)

Campos JH. Lung isolation techniques. Anesthesiol Clin North America 2001;19:455-474.

Campos JH, Massa FC, Kernstine KH. The incidence of right upper-lobe collapse when comparing a right sided double-lumen tube versus a modified left double-lumen tube for left-sided thoracic surgery. *Anesth Analg* 2000;90:535-540.

Cheatham ML, Promes JT. Independent lung ventilation I the management of traumatic bronchopleural fistula. *Am Surg* 2006;72:530-533.

Strange C. Double-lumen endotracheal tubes. Clin Chest Med 1991;12:497-506.

RATIONALE (7)

Answer: A

When a double lumen endotracheal tube cannot be removed at the conclusion of a surgical procedure, it is often beneficial to withdraw the tube until the tip is in the lumen of the trachea. In this patient, frequent coughing might have been an indication that the tip of the endotracheal tube was irritating the carina. The length of the airway from the teeth to the carina is approximately 22-30 cm. In most male and female adults, the endotracheal tube marking should be 22-24 cm at the teeth or gingival ridge in the edentulous patient. The finding of previously undetected right upper lobe atelectasis suggest that the endotracheal tube had migrated into the right mainstem bronchus in the bronchus intermedius and occluded the right upper lobe.

#### **REFERENCES (7)**

- Benumof JL. Separation of the 2 lungs (double-lumen tubes, bronchial-blockers, and endobronchial single-lumen tubes). In Benumof JL, ed. *Airway Management. Principles and Practice*. St. Louis, MO: Mosby; 1996:412-443.
- Campos JH. Lung isolation techniques. Anesthesiol Clin North America 2001;19:455-474.
- Campos JH, Massa FC, Kernstine KH. The incidence of right upper-lobe collapse when comparing a right sided double-lumen tube versus a modified left double-lumen tube for left-sided thoracic surgery. *Anesth Analg* 2000;90:535-540.
- Evron S, Weisenberg M, Harow E, et al. Proper insertion depth of endotracheal tubes in adults by topographic landmarks measurements. *J Clin Anesth* 2007;19:15-19.
- Partridge L, Russell WJ. The margin of safety of a left double-lumen tracheobronchial tube depends on the length of the bronchial cuff and tip. *Anaesth Intensive Care* 2006;34:618-620.
- Stone DJ, Bogdonoff DL. Airway considerations in the management of patients requiring long-term endotracheal intubation. *Anesth Analg* 1992;74:276-287.
- Strange C. Double-lumen endotracheal tubes. Clin Chest Med 1991;12:497-506.

RATIONALE (8)

Answer: B

Pancuronium, vecuronium, and pipecuronium are steroidal neuromuscular blockers that are metabolized in the liver into compounds that also have significant neuromuscular blocking activity and are cleared by the kidney. In patients with renal insufficiency, paralysis may persist well after the neuromuscular blocker has been stopped.

Mivacurium is a short-acting neuromuscular blocker. Its action is stopped by plasma pseudocholinesterase and is not prolonged in hepatic and renal failure. In the ICU it is used mostly for neuromuscular blockade to facilitate brief procedures such as bronchoscopy and central line placement. The action of cisatracurium, an intermediate neuromuscular blocker, is terminated at physiologic pH and temperature by Hofmann degradation to inactive products.

Cisatracurium does not accumulate with renal insufficiency and does not have active metabolites that will prolong neuromuscular blockade.

#### **REFERENCES (8)**

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- Coursin DB, Pearl RG, Prough DS, eds. *Critical Care Medicine*. Philadelphia, PA: Lippincott; 2002:147-167.
- Newman M, Zapol W, eds. Principles of Anesthesia. New York, NY: McGraw-Hill; 2007. In press.
- Pino RM. Residual neuromuscular blockade: a persistent clinical problem. *Intl Anesth Clin* 2006;44:77-90.
- Pino RM, Ali HH. Monitoring and managing neuromuscular blockade. In Longnecker D, Brown D, Segredo V, Caldwell JE, Matthay MA, et al. Persistent paralysis in critically ill patients after long-term administration of vecuronium. *N Engl J Med* 1992; 327:524-528.
- Young CC, Prielipp RC. Sedation, analgesia, and neuromuscular blocking drugs. In Murray MJ, Coursin DB, Pearl RG, Prough DS, eds. *Critical Care Medicine*. Philadelphia, PA: Lippincott; 2002:147-167.

RATIONALE (9)

Answer: E

Established access to the airway is a prerequisite for all percutaneous tracheostomies (PCTs). PCT has no role in the emergent setting. PCTs may be performed in patients with upper airway obstruction, but PCTs should be performed if the patient requires high Fio<sub>2</sub> or significant positive end respiratory pressure (greater than 8-10 cm of H<sub>2</sub>).

Patients with unstable cervical spines due to fractures or ligament injury, and those with limited neck extension, are also not good candidates. Uncorrectable coagulopathy, the presence of aneck mass, and previous neck surgery are also relative contraindications. PCT should not be performed in patients with a history of mediastinal radiation, as fibrosis of the thorax may preclude mobilization of the trachea. Although a previous surgical tracheostomy with a healed stoma is a relative contraindication, some patients with minimal scarring can safely have a PCT performed.

#### **REFERENCES (9)**

Arabi Y, Haddad S, Shirawi N, Al Shimemeri A. Early tracheostomy in intensive care trauma patients improves resource utilization: a cohort study and literature review. Crit Care. 2004; 8:R347-R352.

Berrouschot J, Oeken J, Steiniger L, Schneider D. Perioperative complications of percutaneous dilational tracheostomy. Laryngoscope. 1997 Nov;107(11 Pt 1):1538-1544.

Moe KS, Stoeckli SJ, Schmid S, Weymuller EA Jr. Percutaneous tracheostomy: a comprehensive evaluation. Ann Otol Rhinol Laryngol. 1999; 108:384-391.

Powell DM, Price PD, Forrest LA. Review of percutaneous tracheostomy. Laryngoscope. 1998 Feb;108:170-177.

RATIONALE (10)

Answer: D

Etomidate is an imidazole derivative that offers significant ability as an induction agent to facilitate endotracheal intubation. It provides rapid predictable onset of action and recovery with little effect on cardiovascular stability, limited suppression of ventilation, and lack of histamine release. It does, however, inhibit adrenal mitochondrial hydroxylase activity, resulting in a decrease in cortisol production. Although the effect is usually subclinical, in light of recent reports of relative adrenal insufficiency in septic shock, etomidate should be used with caution in these patients. If etomidate is to be utilized, concomitant administration of corticosteroids should be considered.

#### **REFERENCES (10)**

- Bergen NM, Smith DC. A Review of etomidate for rapid sequence intubation in the emergency department. *J Emerg Med* 1997;15:221-230.
- Deitch S, Davis DP, Schatteman J, et al. The use of etomidate for prehospital rapid-sequence intubation. *Pre-hosp Emerg Care* 2003;7:380-383.
- Horak J, Weiss S. Emergent management of the airway: new pharmacology and the control of comorbidities in cardiac cisease, ischemia, and valvular heart disease. Crit Care Clin 2000;16:411-427.
- Jackson, WL Jr. Should we use etomidate as an induction agent for endotracheal intubation in patients with septic shock? *Chest* 2005;127:1031-1038.

RATIONALE (11) Answer: D

Because oxyhemoglobin saturations are being maintained with bag-mask ventilation, it is appropriate to allow a more experienced intubator the opportunity to attempt to obtain an artificial airway. Even in the presence of adequate bag-mask ventilation saturation, the resident should not attempt a second time. Answers B and E would apply if oral intubation could not be achieved.

#### **REFERENCES (11)**

George E, Haspel KL. The difficult airway. Intl Anesth Clin 2000;38:47-63.

Jaber S, Amraoui J, Lefrant JY, et al. Clinical practice and risk factors for immediate complications of endotracheal intubation in the intensive care unit: a prospective, multiple-center study. *Crit Care Med* 2006;34:2355-2361.

Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesth* 2003;98:1269-1277.

Walz JM, Zayaruzny M, Heard SO. Airway management in critical illness. Chest 2007;131:608-620.

RATIONALE (12)

Answer: A

Residual neuromuscular blockade is a common problem. All of the above choices should be considered when determining if a patient is suitable for extubation. The only direct measurement of muscle strength is the assessment of twitch strength after motor nerve stimulation. The most common method to accomplish this is train-of-four stimulation of the ulnar nerve at the wrist. With train-of-four stimulation, 4 stimuli at 2 Hz are repeated every 10-12 seconds. A comparison of the strength of the first twitch  $(T_1)$  of the adductor pollicis brevis muscle is made 2 seconds after the fourth twitch  $(T_4)$ . When the strength of the first twitch is reduced to 90, 80, and 75% of the maximum twitch height in the absence of neuromuscular blockade, 1, 2, or 3 twitches will be elicited. At a  $T_4/T_1$  ratio of 0.75 (3 twitches), patients will manifest a 5-second head lift, achieve a vital capacity of 15-20 mL/kg, have an effective cough, and can generate an inspiratory force of - 25 cm  $H_2O$ . However, upper esophageal sphincter tone is reduced at  $T_4/T_1$  ratios <90%, with the risk of aspiration.

An inspiratory force of -20 cm H<sub>2</sub>O may be inadequate and is not a firm indicator for sufficient reversal of a neuromuscular blockade to maintain a patent airway. An adequate tidal volume on pressure support ventilation of 16 cm H<sub>2</sub>O may be markedly reduced at lower levels of ventilatory support. Patients may follow commands and have respiratory rates within normal ranges without complete return of muscle function after neuromuscular blockade.

#### **REFERENCES (12)**

- Ali HH. Criteria of adequate clinical recovery from neuromuscular block. *Anesthesiology* 2003;98:1278-1280.
- Eriksson LI. Evidence-based practice and neuromuscular monitoring: it's time for routine quantitative assessment. *Anesthesiology* 2003;98:1037-1039.
- Eriksson LI, Sundman E, Olsson R, et al. Functional assessment of the pharynx at rest and during swallowing in partially paralyzed humans: simultaneous videomanometry and mechanomyography of awake human volunteers. *Anesthesiology* 1997;87:1035-1043.
- Kopman A, Yee P, Neuman G. Relationship of the train-of-four fade ratio to clinical signs and symptoms of residual paralysis in awake volunteers. *Anesthesiology* 1997;86:765-761.
- Pino RM. Residual neuromuscular blockade: a persistent clinical problem. *Intl Anesth Clin* 2006;44:77-90.

RATIONALE (13)

Answer: B

Assessing and describing the level of sedation in a critically ill patient can be difficult. Subjective methods (such as the Sedation-Agitation Scale [SAS] or the Ramsay Scale) and objective methods (heart rate variability, lower esophageal sphincter contractility, or various permutations of the EEG) have been used in efforts to quantify the depth of sedation. Many medications such as propofol, etomidate, thiopental, and benzodiazepines cause a biphasic change in EEG frequency, with lower doses increasing the frequency and higher doses slowing the EEG.

The bispectral index (BIS) is a modified EEG that attempts to overcome this biphasic response to provide a discrete value between 0 and 100. The BIS number for awake patients is in the mid- to upper 90s. Moderate sedation will reduce the BIS to the 70s or 80s, while the BIS during general anesthesia is less than 60. The BIS and similar technologies have sufficient limitations to prevent unambiguous assessments of the depth of sedation. Often, electromyographic activity is the major confounding factor. However, when the patient is chemically paralyzed, such activity is eliminated. A BIS score of 90 indicates that this paralyzed patient is awake.

Opiates, such as hydromorphone, do not alter awareness, in contrast to the sedative and amnestic properties of midazolam. Pressure support ventilation is not an option in this paralyzed patient with abdominal compartment syndrome.

#### **REFERENCES (13)**

- Frenzel D, Greim CA, Sommer C, Bauerle K, Roewen N. Is the bispectral index appropriate for monitoring the sedation level of mechanically ventilated surgical ICU patients? *Intensive Care Med* 2002;28:178-183.
- Nasraway SA, Wu EC, Kelleher RM, Yasuda CM, Donnell AM. How reliable is the Bispectral Index in critically ill patients? A prospective, comparative, single-blind observer study. *Crit Care Med* 2002;30:1483-1487.
- Simons LE, Riker RR, Prato BS, Fraser GL. Assessing sedation during intensive care unit mechanical ventilation with the bispectral index and the sedation-agitation scale. *Crit Care Med* 1999;27:1499-1504.
- Sackey PV, Randell PJ, Granath F, et al. Bispectral index as a predictor of sedation depth during isoflurane or midazolam sedation in ICU patients. *Anaesth Intensive Care* 2007;35:348-356.
- Tonner PH, Paris A, Scholz J. Monitoring consciousness in intensive care medicine. *Best Pract Res Clin Anaesthesiol* 2006;20:191-200.
- Weatherburn C, Endacott R, Tynan P, et al. The impact of bispectral index monitoring on sedation administration in mechanically ventilated patients. *Anaesth Intensive Care* 2007;35:204-208.

RATIONALE (14)

Answer: E

Neuroleptic malignant syndrome is a life-threatening condition that is caused by an alteration of the central dopamine neurotransmission, in contrast to the hypermetabolism of muscle seen in malignant hyperthermia. Psychiatric and postoperative patients who have nausea are at risk. Following laparoscopic procedures, young females are commonly treated for nausea.

Neuroleptic malignant syndrome can be caused by dopamine antagonists, such as prochlorperazine, promethazine, droperidol, haloperidol and metoclopramide, that are commonly used as antiemetics. Symptoms may include psychomotor agitation, rigidity, akinesia, dystonia, fever greater than 38°C (100.4°F), autonomic instability, leukocytosis, and elevated creatine kinase. Moderate elevations in liver function tests are often present. Treatment is the discontinuation of the offending agent, supportive care, and the administration of bromocriptine and/or dantrolene.

The diagnosis of neuroleptic malignant syndrome can be confounded in patients with psychiatric problems. It can be insidious in ICU patients who may have multiple causes of leukocytosis, fever, and agitation.

#### **REFERENCES (14)**

- Adnet P, Gronert GA. Malignant hyperthermia: advances in diagnostics and management. *Current Opinion Anaesth*, 1999;12:353-358.
- Ball C. Unravelling the mystery of malignant hyperthermia. *Anaesth Intensive Care* 2007;35(Suppl): 26-31. Bhanushali MJ, Tuite PJ. The evaluation and management of patients with neuroleptic malignant syndrome. *Neurol Clin* 2004;22:389-411.
- Burkman JM, Posner K, Domino KB. Analysis of the clinical variables associated with recrudescence after malignant hyperthermia reactions. *Anesthesiology* 2007;106:901-906.
- Halliday NJ. Malignant hyperthermia. J Craniofacial Surg, 2003; 14:800-802.
- Reulbach U, Dutsch C, Biermann T, et al. Managing an effective treatment for neuroleptic malignant syndrome. *Crit Care* 2007;11:R4.
- Sheil AT, Collins KA, Schandl CA, et al. Fatal neurotoxic response to neuroleptic medications: case report and review of the literature. *Am J Forensic Med Pathol* 2007;28:116-120.

RATIONALE (15) Answer: C

Dexmedetomidine is an  $\alpha_2$ -agonist that provides sedation without suppressing spontaneous ventilation. Because of the central  $\alpha_2$  effects, opiate requirements are decreased. This drug facilitates patient cooperation during extubation and has also been used for awake fiberoptic intubation. Dexmedetomine permits neurocongnitive testing during awake craniotomies.

The loading dose is  $1 \mu g/kg$  over 10 minutes, followed by a maintenance infusion rate of 0.2-0.7  $\mu g/kg/h$ . There are only modest decreases in arterial pressure, HR, and cardiac output once the drug has been discontinued after a 24 hour infusion. It is approved for 24-hour use or less. With longer infusions, withdrawal symptoms, such as those seen with the abrupt stoppage of clonidine (nervousness, agitation, headaches, and hypertension), may occur. It does not produce ganglionic blockade and has not been associated with QT prolongation. Dexmetedomidine inhibits cortisol synthesis at concentrations ( $10^{-6}$  M) that are higher than the recommended therapeutic concentration ( $<10^{-9}$  M).

#### **REFERENCES (15)**

- Bergese SD, Khabiri B, Roberts WD, et al. Dexmedetomidine for conscious sedatino in difficult awake fiberoptic intubation cases. *J Clin Anesth* 2007;19:141-144.
- Gerkach AT, Dasta JF. Dexmedetomidine: An updated review. Ann Pharmacother 2007;41:245-252.
- Mack PF, Perrine K, Kobylarz E, Schwarts TH, Lien CA. Dexmedetomidine and neurocognitive testing in awake craniotomy. *J Neurosurg Anesthesiol* 2004;16:20-25.
- MacLaren R, Forrest LK, Kiser TH. Adjunctive dexmedetomidine therapy in the intensive care unit: A retrospective assessment of impact on sedative and analgesic requirements, levels of sedation and analgesia, and ventilatory and hemodynamic parameters. *Pharmacotherapy* 2007;27:351-359.
- Maroof M, Khan RM, Jain D, Ashraf M. Dexmedetomidine is a useful adjunct for awake intubation. *Canadian J Anesth* 2005;52:776-777.
- Venn RM, Bryant A, Hall GM, Grounds RM. Comparison between dexmedetomidine and propofol for sedation in the intensive care unit: patient and clinical perceptions. Br J Anaesth 2001;87:684-690.
- Venn RM, Newman PJ, Grounds RM. A phase II study to evaluate the efficacy of dexmedetomidine for sedation in the medical intensive care unit. *Intensive Care Med* 2003;29:201-207.

RATIONALE (16) Answer: C

Succinylcholine, a depolarizing neuromuscular blocker, has many advantages, including its rapid onset of action and its ultra-short duration of neuromuscular blocking activity. However, this drug is associated with several negative effects. In normal patients, succinylcholine administration will result in a serum potassium increase of 0.5-1.0 mEq/L. The administration of succinylcholine to patients with burns, prolonged immobility, crush injuries, and muscular dystrophies will produce life-threatening hyperkalemia, secondary to the up-regulation of extrajunctional receptors for acetylcholine of the skeletal muscle membrane.

Patients with renal insufficiency often have elevated potassium levels, but the extra-junction receptors do not proliferate.

Although succinylcholine will increase intraocular pressure, further damage to the eye with its use during rapid sequence inductions of general anesthesia has not been borne out.

Patients with myasthenia gravis are resistant to succinylcholine.

#### REFERENCES (16)

Martyn JA, Richtsfeld M. Succinylcholine-induced hyperkalemia in acquired pathologic states. Etiologic factors and molecular mechanisms. *Anesthesiology* 2006;104:158-169.

Pino RM, Ali HH. Monitoring and managing neuromuscular blockade. In Longnecker D, Brown D, Newman M, Zapol W, eds. *Principles of Anesthesia*. New York, NY: McGraw-Hill; 2007. In press. Schow AJ, Lubarsky DA, Olson RP, et al. Can succinylcholine be used safely in hyperkalemic patients? *Anesth Analg* 2002;95:119-122.

RATIONALE (17)

Answer: C

This patient developed an arrhythmia as a consequence of underlying severe hyperkalemia. The increase in potassium by the use of succinylcholine was likely more related to her immobility that caused an up-regulation of extrajunctional receptors for acetylcholine rather than her kidney disease.

Meperidine is a synthetic opioid that was first synthesized as an anticholinergic agent with the properties of atropine and papaverine combined. Meperidine is metabolized to normeperidine. This metabolite is proconvulsant and can cause excitation of the central nervous system. By inhibiting the 5-hydroxytrypamine (5-HT) and norepinephrine (NE) uptake mechanisms, it can also cause behavioral, autonomic, and neuromuscular symptoms that are virtually identical to the serotonin syndrome (tremors, salivation, ataxia, spasticity, and convulsions).

When uptake is inhibited with a monoamine oxidase inhibitor (MAOI), the further increase in 5-HT and NE levels, with the added effect of meperidine, may cause sudden severe excitatory effects and death. Traditionally, MAOIs were used as antidepressants. However, they are increasingly being used to treat Parkinson disease.

#### REFERENCES (17)

Fernandez HH, Chen JJ. Monamine oxidase inhibitors: Current and emerging agents for Parkinson disease. *Clin Neuropharmacol* 2007;30:150-168.

Jankovic J. New and emerging therapies for Parkinson disease. Arch Neurol 1999;56:785-790.
 Latta KS, Ginsberg B, Barkin RL. Merperidine: a critical review. Am J Therapeutics 2002;9:53-68.
 Wu RM, Murphy DL, Chiueh CC. Selegiline (l-deprenyl) as a unique neuroprotective agent for chronic neurodegenerative disorders: a lesson from MAO inhibition. Current Medicinal Chemistry 2004;4:255-267.

RATIONALE (18)

Answer: C

The goals during the intubation of this patient are to prevent pulmonary aspiration, maintain cervical spine stability, and minimize the risk of hypercarbia and hypoxemia. Because the patient was admitted after a trauma, a full stomach must be assumed. Fiberoptic-guided intubation requires time to anesthetize the posterior pharynx with local anesthesia and will also blunt any airway reflexes in response to ventilation. Mask ventilation is not indicated in a patient with a full stomach unless it is used as a rescue for a failed intubation. Although the patient's neurologic status has declined, it is unlikely that direct intubation without the use of sedation would be possible.

Cervical collars often prevent the use of a jaw-thrust maneuver that can aid in the visualization of the vocal cords. With in-line manual stabilization, the stability of the cervical spine can be maintained so that the cervical collar can be removed, facilitating the endotracheal intubation.

#### **REFERENCES (18)**

Forster N, Engelhard K. Managing elevated intracranial pressure. *Curr Opin Anaesthesiol* 2004;17:371-376.

Jaber S, Amraoui J, Lefrant JY, et al. Clinical practice and risk factors for immediate complications of endotracheal intubation in the intensive care unit: a prospective, multiple-center study. *Crit Care Med* 2006;34:2355-2361.

Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists *Anesth* 2003;98:1269-1277.

Rosen P, Sloane C, Ban KM, et al. Difficult airway management. *Intern Emerg Med* 2006;1:139-147. Shearer V. Modern airway management for the trauma patient. *Curr Opin Anaesthesiol* 2000;13:135-139. Strawn JR, Keck PE, Caroff SN. Neuroleptic malignant syndrome. *Am J Psychiatry* 2007;164:870-876. Walls RM. Management of the difficult airway in the trauma patient. *Emerg Med Clin NA* 1998;16:45-61.

RATIONALE (19)

Answer: D

Angioedema that can cause a rapid and progressive airway compromise can occur with the use of angiotensin converting enzyme (ACE) inhibitors. ACE inhibitors cause 25-39% of nonhereditary angioedemas. It has been suggested that the incidence of angioedema with lisinopril is greater than with the other ACE inhibitors. The exact pathophysiologic mechanism is unknown, but it may involve inhibition of kinase activity with the accumulation of tissue mediators. Up to 20% of patients may present with acute dyspnea, dysphagia, dysphonia and stridor. As many as 22% of patients with angioedema induced by ACE inhibitors require airway intervention, which may include cricothyroidotomy if attempts at direct visualization of the larynx fail. Once the airway is secured, the angioedema is self-limiting and resolves. There is no evidence that pharmacologic intervention effectively treats this type of angioedema.

#### **REFERENCES (19)**

- Agah R, Bandi V, Guntupalli II. Angioedema: the role of ACE inhibitors and factors associated with poor clinical outcome. *Intensive Care Med* 1997;23:793-796.
- Chin AG, Newkirk KA, Davidson BJ, Burningham AR, Krowiak EJ, Deeb ZE. Angiotensinconverting enzyme inhibitor-induced angioedema: a multicenter review and an algorithm for airway management. *Ann Otol Rhinol Laryngol* 2001;110:834-840.
- Sarkar P, Nicholson G, Hall G. Brief review: angiotensin converting enzyme inhibitors and angioedema: anesthetic implications. *Can J Anesth* 2006;53:994-1003.

RATIONALE (20)

Answer: D

In patients with renal failure, it is essential to use drugs that are not eliminated by the kidney. Morphine is metabolized in the liver to morphine-6-glucuronide, a potent, active metabolite that depends upon renal excretion and will accumulate with renal insufficiency. In contrast, although the metabolite of hydromorphine, hydromorphine-3-glucuronide, is cleared by the kidney, it has no biological activity.

Cisatracurium is a benzylisoquinoline neuromuscular blocker that is degraded in the plasma by Hofmann elimination. It does not depend upon organ elimination for the termination of its action. The steroidal neuromuscular blockers vecuronium and pancuronium are metabolized to the active metabolites, 3-desacetylvecuronium and 3-hydroxpancuronium, respectively. In patients with renal failure, neuromuscular blockade with these drugs will be prolonged.

#### **REFERENCES (20)**

- Chang AK, Bijur PE, Meyer RH, Kenny MK, Solorzano C, Gallagher EJ. Safety and efficacy of hydromorphone as an analgesic alternative to morphine in acute pain: a randomized clinical trial. *Ann Emerg Med* 2006;48:164-172.
- Pino RM, Ali HH. Monitoring and managing neuromuscular blockade. In Longnecker D, Brown D, Newman M, Zapol W, eds. *Principles of Anesthesia*. New York, NY: McGraw-Hill; 2007. In press.
- Segredo V, Caldwell JE, Matthay MA, et al. Persistent paralysis in critically ill patients after long-term administration of vecuronium. *N Engl J Med* 1992;327:524-528.
- van Dorp EL, Romberg R, Sarton E, Bovill JG, Dahan A. Morphine-6-glucuronide: morphine's successor for postoperative pain relief? *Anesth Analg* 2006;102:1789-1797.
- Wittwer E, Kern SE. Role of morphine's metabolites in analgesia: concepts and controversies. *AAPS J* 2006;8:E348-E352.

RATIONALE (21)

Answer: D

Malignant hyperthermia (MH) is an autosomal dominant abnormality in which patients respond ("trigger") to succinylcholine and all volatile anesthetics with a profound hypermetabolism of the skeletal muscles. Rapidly rising hypercarbia is the first sign of MH, followed by a temperature increase of 1-2°C every 5 minutes, skeletal muscle rigidity even in the presence of neuromuscular blockade, tachycardia, and acidosis. Biochemical studies indicate that the primary defect in MH-susceptible patients is the skeletal muscle release channel, called the ryanodine receptor. Through the increased sensitivity of the ryanodine receptors altered by mutation, succinylcholine and the volatile agents (halothane, isoflurane, enflurane, desflurane, and sevoflurane), enhance the rate of calcium release from the sarcoplasmic reticulum.

Death is certain unless treatment is begun with dantrolene. Dantrolene is a skeletal muscle relaxant that acts on the myoneural junction to block calcium release from the sarcoplasmic reticulum. The initial dose of dantrolene is 2.5 mg/kg that is repeated until the symptoms resolve. The maximum dose is 10 mg/kg. A dose of 1 mg/kg can be given every 6 hours for 48-72 hours to prevent the recrudescence of MH. Approximately 50% of all MH episodes have been preceded by 1 or more uneventful anesthetics. An MH episode can be delayed up to 24 hours after surgery.

#### REFERENCES (21)

Adnet P, Gronert GA. Malignant hyperthermia: advances in diagnostics and management. *Current Opinion Anaesth*, 1999;12:353-358.

Burkman JM, Posner K, Domino KB. Analysis of the clinical variables associated with recrudescence after malignant hyperthermia reactions. *Anesthesiology* 2007;106:901-906.

Halliday NJ. Malignant hyperthermia. J Craniofacial Surg, 2003:14:800-802.

Klick JC, Pino RM. Drug Overdose, Poisoning, and Adverse Drug Reactions. In Bigatello LM, et al., eds *Critical Care Handbook of the Massachusetts General Hospital*. Philadelphia, PA: Williams and Willkins; 2006:582-606.

RATIONALE (22)

Answer: A

Current Advanced Cardiac Life Support guidelines recommend the use of capnometry to help confirm the correct placement of the endotracheal tube. Colorimetric capnometry for confirming tracheal intubation outside the operating rooms is commonly used because of its ease of use, portability, and low cost (compared to infrared sidestream or mainstream capnometers). Colorimetric capnometry is based on CO<sub>2</sub> inducing a chemical indicator to change color. The colormetric device is placed between the endotracheal tube and the bag-mask device. As the inspired and expired gases pass through the colorimeter, the chemical indicator changes color, depending on the CO<sub>2</sub> concentration. Most colorimeters can indicate a CO<sub>2</sub> concentration of approximately 0.5% to greater than 2%.

In this case, a low cardiac output is the most likely cause of low levels of detectable end-tidal  $\mathrm{CO}_2$ . Both human and animal studies have shown that decreasing cardiac output causes a concomitant decrease in end-tidal  $\mathrm{CO}_2$ . One study used the concentration of end-tidal  $\mathrm{CO}_2$  as a predictor of the return of spontaneous circulation. Using the colorimetric end-tidal  $\mathrm{CO}_2$  detector, Nakatani and colleagues found that, if end-tidal  $\mathrm{CO}_2$  was persistently less than 0.5% after 7 minutes, the return of spontaneous circulation was 17%. End-tidal  $\mathrm{CO}_2$  results of of 0.5 to 2.0% and >2% were associated with returns of spontaneous circulation of 24% and 48%, respectively. The causes of the low cardiac output during resuscitative efforts could be due to ineffective chest compressions, hypovolemia, tension pneumothorax, or pulmonary embolism.

An esophageal intubation may initially have detectable levels of end-tidal  $\rm CO_2$  due to expired air forced into the stomach during mask ventilation. However, after several positive pressure breaths, the  $\rm CO_2$  would be washed out and end-tidal  $\rm CO_2$  will no longer be detected.

Hyperventilation could cause low levels of end-tidal CO<sub>2</sub> but hypoventilation should increase levels of end-tidal CO<sub>2</sub>. Oxygen flow rates should not make any difference in end-tidal CO<sub>2</sub> unless the manual resuscitator has an incompetent 1-way valve.

#### **REFERENCES (22)**

- Isserles SA, Breen PH Can changes in end-tidal Pco<sub>2</sub> measure changes in cardiac output? *Anesth Analg* 1991; 73:808-814.
- Jin X, Weil MH, Tang W, et al. End-tidal carbon dioxide as a noninvasive indicator of cardiac index during circulatory shock. Crit Care Med 2000; 28:2415-2419.
- Nakatani K, Yukioka H, Fujimori M, et al. Utility of colorimetric end-tidal carbon dioxide detector for monitoring during prehospital cardiopulmonary resuscitation. *Am J Emerg* 1999; 17:203-206.
- Petroianu GA, Maleck WH, Bergler WF, Altmannsberger S, Rufer R. Preliminary observations on the Colibri CO<sub>2</sub>-indicator. *Am J Emerg Med* 1998; 16:677-680.
- Soubani AO. Noninvasive monitoring of oxygen and carbon dioxide. *Am J Emerg Med* 2001; 19:141-146.

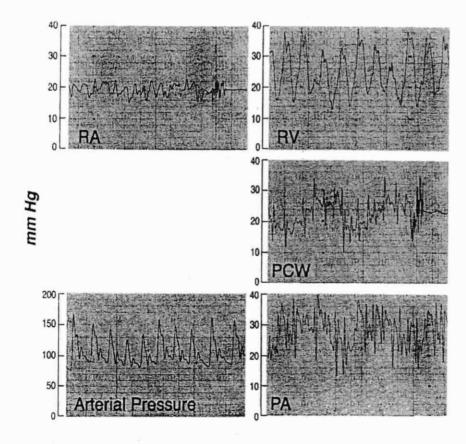
## SECTION 2: Cardiovascular

### **SECTION 2: CARDIOVASCULAR**

Instructions: For each question, select the most correct answer.

1. A 59-year-old female presents to the emergency department with dyspnea and dizziness. She has a recent diagnosis of lung cancer and is being considered for radiation therapy. She was diagnosed with "early pneumonia" 4 days ago by her primary physician and was started on an oral antibiotic as an outpatient. She has a temperature of 38°C (100.4°F), a BP of 86/40 mm Hg, RR 30/min, and HR 115/min. Her physical examination showed significant difficulty in breathing, a mild alteration of mental status, and cool extremities.

At  $16,000 \times 10^3/\mu L$ , her white blood cell count is elevated. Her chest radiograph reveals a perihilar opacity on the right, but no prior chest radiographies are available for comparison. She is brought to the ICU, where her BP fails to improve with additional crystalloid administration. She receives a dopamine infusion to raise the BP to a mean arterial pressure of >65 mm Hg. The working diagnosis is severe sepsis, with sepsis-associated hypotension. A pulmonary artery catheter is inserted, and the following waveforms are observed

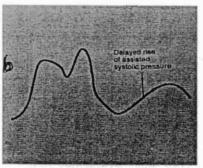


Which of the following is the most appropriate next step?

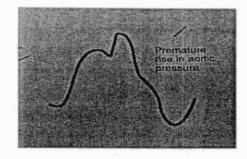
A. Thrombolytics

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- B. Echocardiogram
- C. Cardiac catheterization
- D. Insertion of an intraaortic balloon pump
- 2-3. A 52-year-old male is admitted to the emergency department with an acute ST-segment elevation myocardial infarction, with cardiogenic shock. He is taken to the cardiac catheterization laboratory, where he has a successful balloon angioplasty. After the procedure, the patient is brought to the coronary care unit on a ventilator, and with an intraaortic balloon pump. The following waveform is noted with intraaortic balloon counterpulsation. This waveform indicates which of the following problems with the intraaortic balloon pump?



#2



#3

- 2. A. Late deflation
  - B. Late inflation
  - C. Early deflation
  - D. Early inflation
- 3. A. Late deflation
  - B. Late inflation
  - C. Early deflation
  - D. Early inflation

- 4. A patient suffers an in-hospital cardiac arrest. Of the following, which one has no predictive value with regard to the possibility of surviving to hospital discharge?
  - A. Coronary perfusion pressure during cardiopulmonary resuscitation (CPR)
  - B. End-tidal CO, during CPR
  - C. Neurologic examination findings immediately after return of spontaneous circulation
  - D. Failure to achieve return of spontaneous circulation after 30 minutes of resuscitative efforts
- 5. Which of the following is most likely to have negative effects on coronary perfusion pressure during a cardiopulmonary resuscitation?
  - A. Chest compression rate of 100/min
  - B. Chest compression depth of 1.5-2.0 inches (38-51 mm)
  - C. RR 20/min (after intubation)
  - D. Uninterrupted chest compressions
- 6. A patient is brought to the emergency department with cardiac arrest. The patient had an unknown "down time" but is pulseless on arrival and is being masked ventilated. You perform endotracheal intubation and use colorimetric end-tidal CO<sub>2</sub> testing to help confirm endotracheal tube placement. There is some mild color change on the end-tidal CO<sub>2</sub> detector but not the dramatic color change that is typically associated with successful endotracheal intubation. On auscultation of the chest, breath sounds can be heard with manual ventilation, and no rush of air is heard over the stomach with ventilation. Chest compressions are continued. You ventilate the patient through the endotracheal tube 10 times, and the color change does not go away, but still does not provide the typical degree of color change.

Which of the following statements most likely explains the low-level color variation?

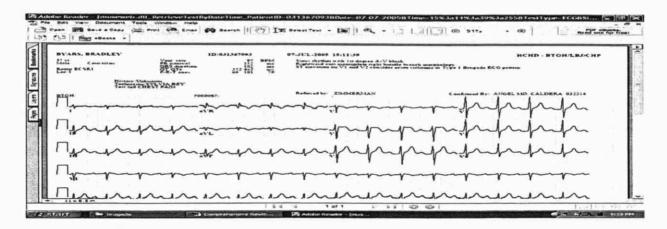
- A. Esophageal intubation
- B. Venous hyperoxia postcardiac arrest
- C. Hypoventilation with the bag ventilation
- D. Low cardiac output
- E. Severe acidosis

7. A 40-year-old male with hypertension is admitted to the ICU for 3 days with right leg swelling. He also noted pain, swelling, and a bluish discoloration. The patient had a previous left leg below-the-knee amputation due to a motor vehicle accident and prior venous thrombosis of the left leg, with the placement of an inferior vena cava filter. Vital signs are as follows: BP 102/52 mm Hg; HR 80/min; RR 28/min; temperature 35.2°C (95.4°F). The right leg was noted to be swollen to the inguinal area with bluish discoloration most prominent in the ankle, foot, and thigh area. Femoral and pedal pulses were palpable but decreased.

Laboratory results included white blood cell count 23,500/mm³; hemoglobin 16.4 g/L; prothrombin time 41.3 seconds; partial thromboplastin time 200 seconds; D-dimer positive; sodium 139 mmol/L, potassium 3.5 mmol/L; CO<sub>2</sub> 16 mmol/L; chloride 102 mmol/L; glucose 107 mg/dL; BUN 26 mg/dL; and creatinine 4.1 mg/dL.

Which one of the following is the most important intervention for treatment of this patient's condition?

- A. Catheter-directed thrombolysis
- B. Operative thrombectomy
- C. Amputation of the right leg
- D. Broad spectrum antibiotics
- E. Low-molecular-weight heparin
- 8. A 37-year-old Laotian male had a sudden cardiac arrest at home. The initial rhythm noted by paramedics was ventricular fibrillation. The patient converted to sinus rhythm with 3 stacked shocks. He was admitted to the critical care unit. He had no known coronary artery disease risk factors and exercised regularly without any complaints of chest pain. He took no medications or illicit drugs. His physical examination was unremarkable, and his first cardiac enzymes were creatinine kinase 400 U/L, creatinine kinase-MB 18 U/L, and troponin I 1.5 µg/mL. His ECG on arrival to the hospital is shown below.



Which of the following interventions should be considered for this patient?

- A. Implantable automatic defibrillator
- B. Initiation of amiodarone
- C. Initiation of a β-blocker
- D. Thrombolysis
- E. Initiation of procainamide
- 9. Which one of the following is most correct concerning recombinant tissue plasminogen activator thrombolytic therapy of acute pulmonary embolism?
  - A. Therapy is contraindicated in a patient with a thrombotic stroke 6 months previously
  - B. Recommended infusion time is 1 hour
  - C. In the patient with profound hypotension, despite high dose vasopressors, administration should be accelerated
  - D. If a pulmonary artery catheter is in place, tissue plasminogen activator should be delivered through the distal port of the catheter

10. A 45-year-old male is admitted to the cardiac intensive care unit in cardiogenic shock after an acute left ventricular myocardial infarction. Systemic BP is 80/40 mm Hg, and HR is 105/min. A pulmonary artery catheter is inserted through the right internal jugular vein. Initial pulmonary artery occlusion pressure (PAOP) is 18 mm Hg; cardiac index 1.8 L/min/m². Dopamine infusion titrated to 20 μg/kg/min increases arterial BP to 105/70 mm Hg, but without improvement in cardiac index.

After intraaortic balloon pump (IABP) placement, the cardiac output increases to 2.5 L/min/m<sup>2</sup> and PAOP decreases to 15 mm Hg. Three hours after the percutaneous coronary angioplasty with stenting, dopamine is titrated off. Overnight, the patient's hemodynamic parameters remain within acceptable limits. Later that morning, liver function tests show an acute increase in alanine aminotransferase and aspartate aminotransferase. The figure below shows the patient's morning IABP tracing.

Which one of the following is the most likely cause of the acute hepatic dysfunction?

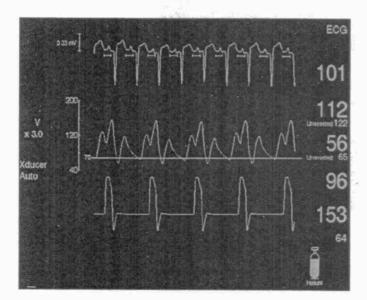
A. Early IABP inflation

B. Every other beat IABP support

C. Late IABP inflation

D.IABP malposition

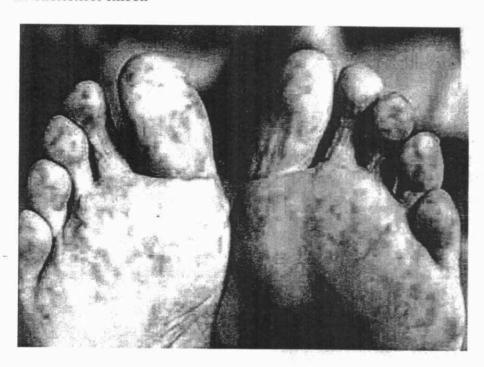
E. Inadequate inflation of the IABP



11. A 66-year-old male with a history of repeated episodes of chest pain and shortness of breath presented with an acute myocardial infarction. The patient underwent thrombolytic and anticoagulant treatments. Cardiac catheterization showed severe 3-vessel coronary artery disease and an ejection fraction of 28%. The patient was scheduled for elective coronary artery bypass graft surgery. One day after the angiographic procedure, the patient complained of diffuse muscle pain and was found to have a serum creatinine of 2.8 mg/dL (previously it was 1.2 mg/dL) and discoloration of his feet (see the photograph below).

Which one of the following is the cause of his problem?

- A. Reaction to radiocontrast
- B. Bleeding from anticoagulation after thrombolytic therapy
- C. Dissecting aortic aneurysm
- D. Postinfarction congestive heart failure
- E. Cholesterol emboli



12. 60-year-old male is admitted to the ICU after cardiac surgery. Physical examamination and chest radiography are consistent with pulmonary edema. A vasoactive drug is administered.

Parameters	On admission	After therapy
BP (mm Hg)	98/66	88/50
Mean right atrial pressure (mm Hg)	23	15
Pulmonary artery pressure (mm Hg)	48/26	40/20
Pulmonary artery occlusion pressure (mm Hg)	26	18
Cardiac index (L/min/m²)	1.6	2.8
Heart rate (beats/min)	110	119

Which one of the following drugs was administered?

- A. Nitroglycerin
- B. Milrinone

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- C. Dopamine
- D. Norepinephrine

13. A 60-year-old man with severe ischemic cardiomyopathy presents with profound dyspnea. The patient is found to have a pulmonary edema secondary to the decompensated heart failure. He is given supplemental oxygen with continuous positive airway pressure, and is admitted to the ICU. A pulmonary artery catheter is inserted to help guide therapy, and the initial data confirms a diagnosis of high pressure pulmonary edema and biventricular failure. A single continuous infusion is started. Thirty minutes after starting the infusion, the oxygen requirement has decreased and the following changes are seen:

CO PAOP SVR HR 
$$\uparrow$$
  $\downarrow\downarrow$   $\downarrow\downarrow$  =

CO = cardiac output

PAOP = pulmonary artery occlusion pressure

SVR = systemic vascular resistance

HR = heart rate

Which one of the following drugs is being infused?

- A. Bumetanide
- B. Nesiritide (brain natriuretic peptide)
- C. Nitroglycerin
- D. Epinephrine
- E. Dopamine (10 μg/kg/min)

14. A 54-year-old male is admitted to the emergency room with severe substernal chest pain and acute shortness of breath. The patient has a history of non-insulin-dependent diabetes mellitus and hypertension controlled with medications. On examination, the patient is diaphoretic and in respiratory distress. Vital signs reveal a HR of 120/min, BP of 95/50 mm Hg, and a RR of 24/min. Lung examination reveals bilateral crackles; the heart examination is significant for regular tachycardia, with normal S1 and S2. A S3 is present without murmurs or rubs. Chest radiography shows changes consistent with a pulmonary edema. A 12-lead ECG is significant for T-wave inversions in V1-V5. Patient is taken emergently for cardiac catheterization. The following hemodynamic values are obtained:

BP	85/46 mm Hg
Right atrium	12 mm Hg
Right ventricle	34/10 mm Hg
Pulmonary artery	34/22 mm Hg
Pulmonary artery (mean)	25 mm Hg
Pulmonary artery occlusion pressure	20 mm Hg
Cardiac output	4 L/min
Cardiac input	2.2 L/min

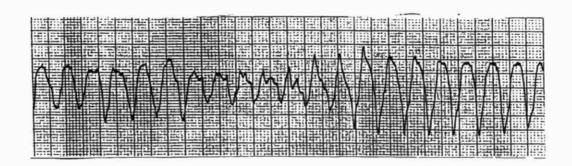
Oxygen consumption is calculated at 180 mL/min/m<sup>2</sup>.

Significant triple vessel disease with a lesion in the proximal left anterior descending artery is found. An intra-aortic balloon pump is placed in preparation for an emergent coronary artery bypass graft.

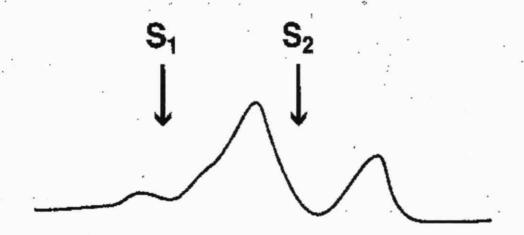
Which one of the following hemodynamic parameters would be most consistent with an effective intra-aortic balloon counterpulsation?

	BP (mm Hg)	PCWP (mm Hg)	CI (mm Hg)	Oxygen Consumption (mL/min/m²)
A.	80/40	20	2.8	190
B.	85/46	16	2.8	200
C.	95/60	16	2.8	140
D.	90/60	22	2.2	180

15. What is the most appropriate initial medication for the rhythm shown in the figure below?

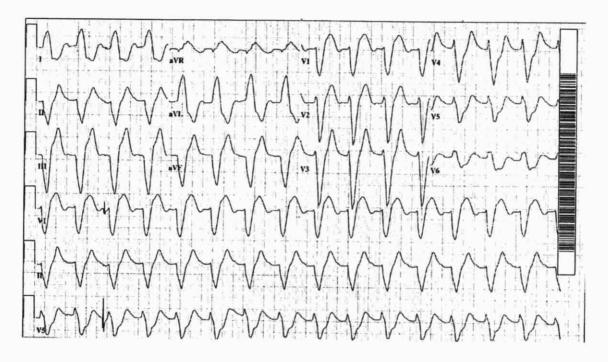


- A. Amiodarone
- B. Verapamil
- C. Quinidine
- D. Magnesium sulfate
- E. Calcium carbonate, insulin+ glucose
- 16. Which one of the following is the most likely diagnosis of a young female presenting with dyspnea on exertion and paroxysmal nocturnal dyspnea? Her arterial pressure waveform is shown in the figure below.



- A. Aortic regurgitation
- B. Dilated cardiomyopathy
- C. Aortic stenosis
- D. Pulmonic regurgitation
- E. Hypertrophic cardiomyopathy

17. A 60-year-old female presents to the emergency department with a diagnosis of acute respiratory failure from severe pneumonia. The patient has a past medical history of end stage renal disease (on hemodialysis), obesity, hypertension, and has been wheelchair-bound for several weeks. A decision is made to intubate the patient and initiate mechanical ventilation. The patient is given midazolam, fentanyl, and succinylcholine. During intubation, a change in her rhythm is noted, as shown in the ECG below.

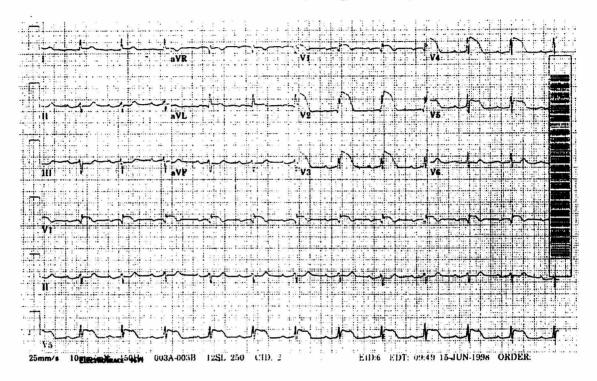


Which of the following is most appropriate therapy at this point?

- A. Cardioversion
- B. Amiodarone
- C. Lidocaine
- D. Calcium Chloride
- E. External pacing with transcutaneous patches

18. A 50-year-old male with a past medical history significant for hypertension presents to the emergency department with complaints of severe substernal chest pain and shortness of breath that started 30 minutes ago while eating at a nearby restaurant.

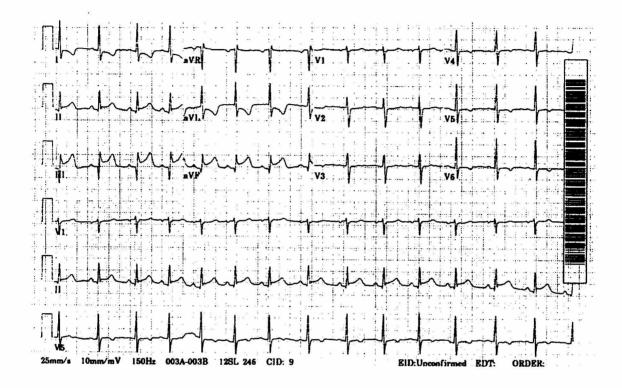
On physical examination, the patient is diaphoretic. Vital signs include a BP of 90/50 mm Hg, HR of 75/min, and RR of 28/min. Physical examination reveals irregular tachycardia with no murmurs and a positive S3. Lung auscultation reveals crackles in both bases. The rest of his examination is unremarkable. An ECG is obtained (see figure below).



Which of the following management options is most appropriate at this point?

- A. Aspirin, IV nitroglycerin, low-molecular-weight heparin, and  $\beta$ -blockers
- B. Aspirin, low-molecular-weight heparin,  $\beta$ -blockers, and glycoprotein IIb/IIIa inhibition
- C. Aspirin and urgent catheterization
- D. Aspirin, β-blockers, and r-TPA
- E. Aspirin, low-molecular-weight heparin, glycoprotein IIb/IIIa inhibition, and placement of intraaortic balloon pump

19. A 60-year-old male presented to the emergency department after experiencing chest pain and shortness of breath for approximately 3 hours. The pain was described as substernal with radiation to his jaw. Past medical history includes hypertension, non-insulin-dependent diabetes mellitus, and hyperlipidemia. On physical examination, his vital signs were as follows: HR 95/min, BP 100/70 mm Hg, and RR 20/min. Patient has increased jugular venous distention, his lungs were clear on auscultation, and his heart sounds were regular with no murmurs, rubs, or gallops. An ECG was obtained (see figure below).



The patient started receiving oxygen via nasal cannula. He was given aspirin, IV heparin, and was started on IV nitroglycerin at a rate of  $10 \,\mu g/min$ . The patient complained of feeling dizzy. His BP dropped to  $75/50 \,mm$  Hg, and his chest pain intensified. Nitroglycerin was stopped and an IV fluid bolus with normal saline solution was given.

Which of the following hemodynamic parameters would be most consistent with this clinical case?

	CVP (mm Hg)	PAP (mm Hg)	PAOP (mm Hg)	CI (L/min/m <sup>2</sup> )
A.	14	25/14	14	2.0
В.	12	35/15	10	1.8
C.	10	30/22	20	1.8
D.	2	15/8	6	1.5
E.	6	20/10	10	3.2

20. A 32-year-old man is admitted to the ICU with a clinical diagnosis of meningococcemia and septic shock. He was intubated in the emergency department for airway protection. Results of a lumbar puncture are compatible with bacterial meningitis. An IV of penicillin is initiated. The chest radiograph shows no abnormalities.

On physical examination, his temperature is 39°C (102.2°F), with a HR of 115/min. An arterial line is inserted, and his mean arterial pressure is 64 mm Hg with the administration of IV norepinephrine. He is sedated and mechanically ventilated. Pulse oximetry is 95%, with a Fio<sub>2</sub> of 0.5 and a positive end expiratory pressue of 5 cm H<sub>2</sub>O.

Since presenting to the hospital (4 hours ago) he has received 2.5 L normal saline solution. His urine output has decreased and is currently 0.25 mL/kg/h over the last 2 hours. He has a left subclavian central line that shows a central venous pressure of 6 mm Hg and a measured Scvo<sub>2</sub> of 60%. White blood cell count is 23,500 x  $10^3$ /µL, hematocrit is 28%, and platelet count is 42,000 x  $10^3$ /µL at the time of the lumbar puncture).

Which of the following therapeutic interventions is most appropriate at this time?

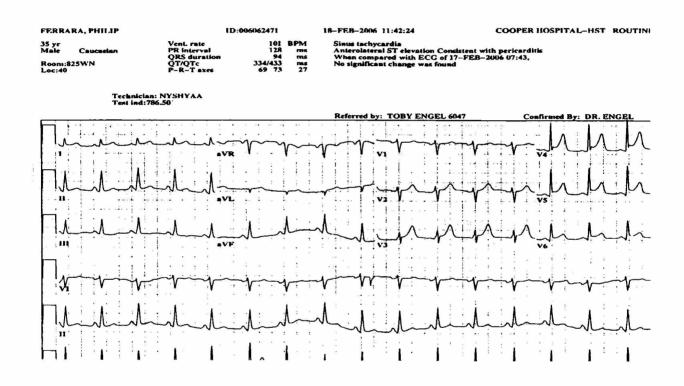
- A. Transfuse 1 U of platelets
- B. Increase norepinephrine to achieve a mean arterial pressure >75 mm Hg
- C. Initiate dobutamine at 5 µg/kg/min
- D. Transfuse 2 U of packed red blood cells
- E. Administer a 1,000-mL bolus of normal saline solution
- 21. A 45 year-old female with no significant past medical history is admitted to the hospital for abdominal pain and weight loss and undergoes a CT scan of the abdomen and pelvis with an IV contrast. After administration of the contrast material, the patient complains of throat tightness, diffuse itching, and feeling light-headed.

Physical examination reveals a diaphoretic and anxious patient. Vital signs are remarkable for a BP of 72/48 mm Hg, HR of 124/min, and RR of 25/min. On examination of the chest, there is diffuse wheezing. A rash is noted on the patient's chest and upper extremities.

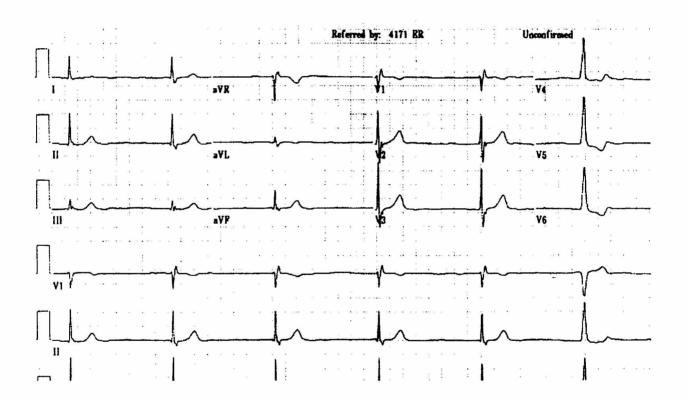
Which of the following is the most appropriate immediate treatment for the patient's hypotension?

- A. Dobutamine
- B. Dopamine
- C. Epinephrine
- D. Corticosteroids
- E. Vasopressin

- 22. You are asked to evaluate a 32-year-old male who presents to the emergency department with complaints of acute onset retrosternal chest pain and shortness of breath. Vital signs are are follows: temperature 37.9°C (100.2°F), HR 110/min, BP 138/85 mm Hg, and RR 20/min. An ECG is obtained and is shown below. Which of the following diagnostic tests would be most appropriate for this case?
  - A. Cardiac catheterization
  - B. CT scan of the chest with IV contrast (pulmonary embolism protocol)
  - C. Transthoracic echocardiogram of the heart
  - D. Lower extremity Doppler ultrasound



- 23. A 72 year-old female at an assisted living facility is brought to the hospital after developing an altered mental status and shortness of breath. The patient has a history of hypertension and chronic osteoarthritis. On examination, her BP is 70/48 mm Hg; HR 30/min, and RR 22/min. The patient is confused and has no focal neurologic findings. Laboratory data show potassium levels of 5.5 mEq/L and creatinine 2.2 mg/dL. An ECG is shown below. Which of the following therapeutic interventions is most appropriate for this patient?
  - A. Atropine
  - B. Calcium chloride
  - C. Transcutaneous pacemaker
  - D. Hemodialysis
  - E. IV pacemaker



- 24. As an immediate effect (during a single breath), which one of the following would increase left ventricular stroke volume?
  - A. Positive pressure expiration
  - B. Relief of inferior vena cava constriction
  - C. Abdominal compression
  - D. Right decubitus positioning
- 25. Which one of the following is most correct concerning vasopressin?
  - A. With the onset of septic shock, vasopressin levels are normal, then rise slowly, typically peaking at 48-72 hours
  - B. The administration of vasopressin in healthy individuals produces similar vasopressor effects as in septic shock
  - C. Vasopressin increases platelet count
  - D. Vasopressin decreases cardiac output at doses higher then 0.04 U/min, primarily due to negative chronotropic effect
- 26. A 55-year-old male presents to the emergency department with hypoxemia, hypotension, and no past medical history of cardiopulmonary disease. He has non-insulin-dependent diabetes mellitus (NIDDM) and chronic renal insufficiency with a creatinine of 2.2. Chest radiograph is without an infiltrate, and his lung examination is normal. His BP initially normalizes with a fluid bolus, but then decreases, requiring 10 μg/kg/min of dopamine to maintain a systolic BP of 90-100 mm Hg. Oxygenation is good with 5 L nasal cannula oxygen.

An echocardiogram reveals a dilated right atrium and right ventricle without any other abnormality. A central venous pressure (CVP) catheter is inserted in the right neck and reveals a CVP of 12 mm Hg and a ScvO2 saturation of 60%. CT scanning and remote interpretation are immediately available. Leg ultrasound, perfusion lung scanning, and angiography are not available in house, but are available by call-in.

Which one of the following is the most appropriate management of this patient?

- A. Dobutamine and thrombolytic therapy
- B. Fluid bolus and thrombolytic therapy
- C. Dobutamine and helical chest CT scan
- D. Fluid bolus and helical chest CT scan
- E. Develop plan based on call-in services

27. A 56-year-old female being treated for unresectable lung cancer is admitted to the ICU with hypotension that is poorly responsive to fluid resuscitation, leading to initiation of vasopressors (dopamine 10μg/mg/min) to maintain adequate blood pressure. A pulmonary artery catheter is inserted and reveals equalization of pressures (right atrial, right ventricular diastolic pulmonary artery occlusive pressure). Emergent echocardiogram reveals right atrial and right ventricular collapse with systole. Cardiac index is 1.0 L/min/m². There is a large 2.5 cm circumferential pericardial effusion present.

Emergent pericardial centesis is performed with removal of approximately 60 µg of fluid. Blood pressure rises significantly, and the patient is weaned from dopamine. Cardiac index increases. The patient is transferred to the cardiac catheterization laboratory. Right atrial mean pressure is 31 mm Hg with an intrapericardial pressure of 31 mm Hg. 800 µg of sanguinous fluid are removed from the pericardial space, and a pig-tail catheter is left to drainage. Right atrial mean pressure is reduced to 23 mm Hg, while the intrapericardial pressure decreases to 8 mm Hg. Electrocardiography confirms that most of the fluid has been removed. Patient is now slightly hypertensive and not receiving vasoactive drugs.

In addition to hemodynamically significant pericardial effusion, which one of the following is also likely to be present?

- A. Primary pulmonary artery hypertension
- B. Secondary pulmonary artery hypertension
- C. Right ventricular infarction
- D. Right to left intracardiac shunt
- E. Effusive-constrictive pericarditis

28. A patient with a known hypertrophic cardiomyopathy and dynamic left ventricular outflow tract obstruction is intubated for community-acquired pneumonia. Urine output is minimal, with no response to a fluid bolus. Patient is tachycardic and has a blood pressure of 118/74 mm HG and a HR 120/min.

Which one of the following therapies is most appropriate as part of the treatment?

- A. Furosemide
- B. Inotropic dose dopamine
- C. Beta blockade
- D. Nitroglycerin
- E. Nicardipine

29. A 42-year-old male with a history of severe dilated cardiomyopathy (ejection fraction 10-15%) presents to the emergency department after a syncopal event. On arrival at the emergency department, he is conscious and alert. While waiting in the triage area to be seen by a physician; he becomes unresponsive. He is rushed into a treatment area, where he is found to be nonbreathing and pulseless. The cardiac monitor shows ventricular fibrillation.

He is promptly intubated; advanced cardiac life support protocols are initiated. After a few minutes, return of spontaneous circulation is achieved. The vital signs at that time are as follows: HR 110/min, RR 20/min (mechanically ventilated), oxygen saturation 100% (high flow supplemental oxygen). The total estimated down time was 5-7 minutes.

After checking a portable chest radiograph to confirm the adequacy of the endotracheal tube placement, which one of the following should be done next?

- A. Stat echocardiogram
- B. Temporary transvenous pacemaker
- C. Pulmonary artery catheter insertion
- D. Administration of magnesium sulfate (MgSO<sub>4</sub>)
- E. Administration of cooling blankets and cold IV fluids
- 30. Which of the following is an expected (ie, normal) change in cardiopulmonary physiology associated with third trimester pregnancy?
  - A. Increased venous return to the heart in the supine position
  - B. Increased pulmonary artery occlusion pressure
  - C. Decreased total circulating blood volume
  - D. Decreased systemic vascular resistance
  - E. Decreased heart rate

- 31. Which of the following statements regarding the period of hemodynamic instability immediately following cardiac surgery (utilizing cardiopulmonary bypass) is most correct?
  - A. Estimation of cardiac preload using measurements of cardiac filling pressure may be challenging due to dynamic changes in myocardial compliance
  - B. Brisk urine output is a good indicator of adequate cardiac preload
  - C. Routine administration of protamine (given after discontinuation of cardiopulmonary bypass) has no known effects on hemodynamics
  - D. If pulseless electrical activity occurs, the patient must be brought back to the operating room immediately in order to relieve cardiac tamponade
- 32. Which of the following statements about post-operative bleeding in the mediastinal and pleural tubes immediately following cardiac surgery is most correct?
  - A. The most common etiology of excessive bleeding is a surgical (technical procedure-related) hemostasis problem
  - B. Thrombocytopenia is uncommon
  - C. Platelet dysfunction is common and typically secondary to hypothermia or cardiopulmonary bypass
  - D. More than 200 mL of blood output in the mediastinal tubes per hour for 4 consecutive hours is an indication for echocardiography

# **SECTION 2: CARDIOVASCULAR**

#### **ANSWERS:**

1-B; 2-D; 3-A; 4-C; 5-C; 6-D; 7-A; 8-A; 9-C; 10-D; 11-E; 12-B; 13-B; 14-C; 15-D; 16-B; 17-D; 18-C; 19-B; 20-E; 21-C; 22-C; 23-B; 24-A; 25-D; 26-D; 27-E; 28-C; 29-E; 30-D; 31-A; 32-C

RATIONALE (1) Answer: B

These waveforms show an equalization of pressures. Note that the diastolic pressures for the right atrium, right ventricle, pulmonary artery and pulmonary capillary wedge pressure are all within 5 mm Hg from each other. Bedside echocardiography is warranted to rapidly evaluate the possibility of a pericardial tamponade.

Thrombolysis is not indicated, as there is neither indication of an acute myocardial infarction in this patient, nor is there any supporting evidence of an acute pulmonary embolism in these waveforms. A trip to the cardiac catheterization laboratory would likely result in the identification of the problem as pericardial tamponade, but the patient is too unstable to be transported, and a coronary intervention is not the expected therapy to solve this problem.

# **REFERENCES (1)**

•

- Al-Hazzouri A, Mazzone P. Hypotension in the intensive care unit. Cleve Clin J Med. 2006; 73:1091-1097.
- Humphreys M. Pericardial conditions: signs, symptoms and electrocardiogram changes. *Emerg Nurse*. 2006; 14:30-36.
- Little WC, Freeman GL. Pericardial disease. Circulation. 2006; 113:1622-1632.
- Pepi M, Muratori M. Echocardiography in the diagnosis and management of pericardial disease. *J Cardiovasc Med.* 2006; 7:533-544.
- Roy CL, Minor MA, Brookhart MA, Choudhry, NK. Does this patient with a pericardial effusion have cardiac tamponade?. *JAMA*. 2007; 297:1810-1818.
- Seferovl PM, Risti AD, Imazlo M, et al. Management strategies in pericardial emergencies. *Herz*. 2006; 31:891-900.

RATIONALE (2) and (3) 2-D; 3-A

There are two types of intraaortic balloon pump mistiming that are potentially deleterious: early inflation and late deflation. Early inflation (question #2) will result in an increased afterload in the late portion of systole. This can reduce cardiac output and increase afterload on an ischemic heart. Late deflation (question #3) will cause an increased afterload in early systole and can also reduce cardiac output.

Other mistiming may make the intraaortic balloon counterpulsation inefficient or not helpful, but these are the two waveforms that represent potentially serious deleterious effects. An example of a proper intraaortic balloon counterpulsation waveform is shown in Figure 1 below.

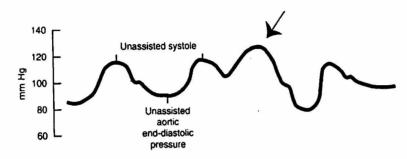


Figure 1. A normal aortic pressure waveform with intraaortic balloon counterpulsation augmentation indicated by the arrow, which corresponds to diastolic augmentation. Reproduced with permission from Kelly RF. Intraaortic balloon counter pulsation. In: Parrillo JE, Dellinger RP, eds. *Principles of Diagnosis and Management in the Adult: A Self Assessment*. Philadelphia, PA: Mosby; 2005.

The balloon pump will inflate in diastole to provide diastolic augmentation of the blood flow, with an increase in coronary artery perfusion pressure. The deflation in systole will decrease afterload and is expected to augment cardiac output. Typically, the intraaortic balloon pump will automatically synchronize with the ECG, but mistiming can still occur and must be recognized.

# **REFERENCES (2 and 3)**

Kelly RF. Intraaortic balloon counter pulsation. In: Parrillo JE, Dellinger RP, eds. *Principles of Diagnosis and Management in the Adult: A Self Assessment*. Philadelphia, PA: Mosby; 2005.

RATIONALE (4)

Answer: C

Although chances of surviving to hospital discharge may be low in many cases of in-hospital cardiac arrest, the chances of survival to discharge is dismal in a few scenarios. Prolonged (>30 min) resuscitation efforts (as in answer D) likely confers no benefit if the return of spontaneous circulation has not been achieved. Failure to increase the end-tidal CO<sub>2</sub> to exceed 8 mm Hg indicates that a markedly low cardiac output is present, and cardiopulmonary resuscitation is not likely to be effective. In a study placing central monitors in subjects receiving chest compressions, a failure to augment coronary artery perfusion pressure to >15 mm Hg identified subjects that could not be resuscitated.

Neurologic findings, including pupillary response, reflexes, and motor activity, may change over the first 72 hours after return of spontaneous circulation. However, at the end of 72 hours, the neurologic findings are highly predictive of neurologic outcome. If the initial rhythm of the cardiac arrest was ventricular fibrillation (and possibly with other initial rhythms of cardiac arrests as well), and the return of spontaneous circulation can be achieved, therapeutic hypothermia should be initiated immediately in order to minimize neurologic injury and give the patient the best chance for a better neurologic outcome.

# **REFERENCES (4)**

Fischer C, Luaute J, Nemoz C, et al. Improved prediction of awakening or nonawakening from severe anoxic coma using tree-based classification analysis. *Crit Care Med* 2006;34:1520-1524.

Paradis NA, Martin GB, Rivers EP, et al. Coronary perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. *JAMA* 1990; 263:1106-1113.

Puttgen HA, Geocadin R. Predicting neurological outcome following cardiac arrest. *J Neurol Sci* 2007 (In print).

Zandbergen EG, de Haan RJ, Stoutenbeek CP, et al. Systematic review of early prediction of poor outcome in anoxic-ischaemic coma. *Lancet* 1998; 352:1808-1812.

RATIONALE (5)

Answer: C

Recent investigations have identified that the quality of cardiopulmonary resuscitation (CPR), in both in-hospital and out-of-hospital cardiac arrests, does not meet consensus recommendations for providing CPR. These studies identified several opportunities to improve the quality of CPR, in both in-hospital and out-of-hospital cardiac arrest, by focusing on adherence to the recommendations for the rate of chest compressions, depth of chest compressions, minimization of "no-flow" periods when chest compressions are interrupted, and avoidance of over-ventilating the patient. Current consensus recommendations advocate a chest compression rate of 100/min. The depth of compressions is recommended to be 1.5-2 inches.

Overventilating the cardiac arrest patient has been reported and can be deleterious. The recommendations are for ventilations to be provided after every 15 chest compressions before intubation, and 12 ventilation/min after the patient is intubated.

#### **REFERENCES (5)**

- 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation* 2005;112(Suppl):IV1-203.
- Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during inhospital cardiac arrest. *JAMA* 2005; 293:305-310.
- Aufderheide TP, Sigurdsson G, Pirrallo RG, et al: Hyperventilation-induced hypotension during cardiopulmonary resuscitation. Circ J Am Heart Assoc 2004; 109:1960-1965.
- Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus of Science. Dallas, TX: American Heart Association; 2000.
- Wik L, Kramer-Johansen J, Myklebust H, et al. Quality of cardiopulmonary resuscitation during outof-hospital cardiac arrest. *JAMA* 2005; 293:299-304.

RATIONALE (6)

Answer: D

Qualitative capnometry is a rapid and simple confirmatory test for endotracheal tube placement. The chemical indicator in the device will change color depending on the carbon dioxide concentration. A quantitative end-tidal CO<sub>2</sub> detector is typically not necessary. In patients with profoundly low cardiac output, end-tidal CO<sub>2</sub> may be difficult to detect because the lungs are not being perfused. A profoundly low cardiac output could cause this clinical scenario, and this may be due to ineffective chest compressions with cardiopulmonary resuscitation, tension pneumothorax, or some other cause of an inadequate cardiac output during CPR.

An esophageal intubation could potentially give a positive reading with a colorimetric indicator because of the air that could be in the stomach when the patient is mask ventilated prior to intubation. However, that color change should go away after delivery of several breaths. This low-level color change persisted, indicating that the esophageal intubation is an unlikely source.

Venous hyperoxia has been demonstrated postcardiac arrest with the return of spontaneous circulation, and is presumed to be secondary to bioenergetic failure and defects in oxygen utilization after a prolonged "down time." This would have no impact on end-tidal CO<sub>2</sub> detection.

# **REFERENCES (6)**

Jin X, Weil MH, Tang W, et al. End-tidal carbon dioxide as a non-invasive indicator of cardiac index during circulatory shock. *Crit Care Med* 2000; 28:2415-2419.

Rivers EP, Rady MY, Martin GB, et al. Venous hyperoxia after cardiac arrest: characterization of a defect in systemic oxygen utilization. *Chest* 1992; 102:1787-1793.

RATIONALE (7)

Answer: A

This patient has phlegmasia cerulea dolens, an infrequent but severe manifestation of venous thrombosis. This condition can result in venous gangrene, arterial compromise, loss of limb, and even death. The most appropriate intervention to treat the underlying condition is catheter-directed thrombolysis to rapidly remove the thrombus and restore venous drainage. There are no well-designed studies evaluating this approach, and it should be reserved for limb salvage after an assessment of the risk/benefit ratio, as compared with routine anticoagulation.

Operative thrombectomy is an alternative intervention, but, due to its high mortality rate, it is usually used after the failure of anticoagulation and thrombolytic therapy.

Although anticoagulation is indicated, heparin will not alleviate the venous obstruction rapidly enough.

While amputation may be needed when other interventions fail, it is reasonable to utilize other interventions to salvage the limb prior to considering amputation.

Although this patient has an elevated white blood cell count and hypothermia, these manifestations are most likely secondary to the tissue ischemia and subsequent inflammatory response, rather than an established infection. It is reasonable to obtain culture data and consider antibiotic therapy, but these interventions would not address the underlying cause.

In some case reports, phlegmasia cerulea dolens has been associated with prior placement of inferior vena cava filters. It may occur in lower as well as upper extremities. With mild manifestations and subocclusive thrombosis, systemic anticoagulation may be sufficient. With occlusive thrombosis and vascular compromise, catheter-directed thrombolysis would be the treatment of choice. In some cases, catheter access to the thrombosis may be impossible and a thrombectomy would be indicated. Fasciotomy may be required after reestablishment of the flow and stabilization of the patient to relieve the elevated compartment pressures.

# **REFERENCES (7)**

- Buller HR, Agnelli G, Hull RD, et al. Antithrombiotic therapy for venous thromboembolic disease, the seventh ACCP conference on antithrombiotic and thrombolytic therapy. *Chest* 2004; 126:401S-428S.
- Centeno RF, Nguyen AH, Ketterer C, et al. An alternative approach: antigrade catheter- directed thrombolysis in a case of phlegmasia cerulea dolens. *Am Surg* 1999; 65:2229-2231.
- Martyn JA, Richtsfeld M. Succinylcholine-induced hyperkalemia in acquired pathologic states: etiologic factors and molecular mechanisms. *Anesthesiology* 104:158, 2006.
- Pino RM, Ali HH. Monitoring and managing neuromuscular blockade. In: Longnecker D, Brown D, Newman M, Zapol W. *Principles of Anesthesia*. New York, NY: McGraw-Hill, 2007, in press.
- Schow AJ, Lubarsky DA, Olson RP, et al. Can succinylcholine be used safely in hyperkalemic patients? Anesth Analg 95:119, 2002.

RATIONALE (8)

Answer: A

This patient's clinical history and ECG findings are typical for Brugada syndrome, which is characterized by syncope, ventricular fibrillation, or sudden death in patients with structurally normal hearts. The ECG pattern is distinctive, with ST-segment elevations in V1-V3 with a normal QT interval. An implantable defibrillator should be considered to prevent recurrent ventricular fibrillation and sudden death.

The syndrome is usually noted in males in the third and fourth decades of life and was first described in Asian men with sudden death. It may be hereditary, with an autosomal dominant pattern of inheritance. A mutation in a cardiac sodium channel  $\alpha$ -subunit gene, SCN5A, has been identified in 15 to 30% of patients.

In patients resuscitated from sudden death with normal ECGs, infusion of type IA antiarrhythmics, such as procainamide, may elicit classic ECG findings. Brugada-type ECGs have also been identified with an overdose of psychotropic drugs.

β-blockers and amiodarone are not helpful in this syndrome and should be avoided. Although thrombolysis could be considered with the ST-segment elevation and sudden death, the classic ECG findings and lack of risk factors suggests other approaches.

#### **REFERENCES (8)**

Brugada P, Brugada J, Brugada R. The Brugada syndrome. Cardiovasc Drugs Ther 2001; 15:15-17. Littmann L, Monroe MH, Kerns WP, et al. Brugada syndrome and "Brugada sign": clinical spectrum with a guide for the clinician. Am Heart J 2003; 145:768-778.

Naccarelli GV, Anzelevitch C, Wolbrette DL, Luck JC. The Brugada syndrome. *Curr Opin Cardio* 2002; 17:19-23.

RATIONALE (9)

Answer: C

In the unstable patient at eminent risk of death, the traditional 2-hour administration period of 100 mg recombinant tissue plasminogen activator (TPA) should be shortened. This is a consensus recommendation without a high level of evidence, but because most patients will either live or die within a short period of time, a short infusion period is clinically logical. One proposed alternative to the traditional 2-hour infusion which seems reasonable is 40 mg over 5 minutes followed by 60 mg over the remainder of the 2-hour period.

Thrombolytic therapy is contraindicated if an acute cerebral vascular event or procedure has occurred within the last 2 months, not 6 months. In most circumstances, an angiographic or CT scan diagnosis of pulmonary embolism (PE) should be obtained prior to thrombolytic therapy. But in patients where the clinical presentation and limited testing (such as echocardiography or leg ultrasound) strongly support the diagnosis, and the transport risk or risk of imminent death is high, thrombolytic therapy may be given without a definitive diagnosis of PE.

Intracranial bleeding is the primary concern with thrombolytic therapy and occurs in 1-2% of patients with PE who are treated with thrombolytic therapy. The primary indication for thrombolytic therapy remains a vasopressor requirement to maintain BP or organ perfusion. Refactory life-threatening hypoxemia, which is rarely seen, would also be an indication. There is some controversy as to whether right ventricular dysfunction in the absence of hemodynamic instability is an indication for thrombolytic therapy; there are numerous conflicting reports in this area. Infusion of a thrombolytic agent directly onto an acute pulmonary arterial thrombus has never been shown to be superior to infusion of the agent through a peripheral vein, since the benefit is due to the systemic fibrinolysis.

### **REFERENCES (9)**

- Firdose R, Elamin EM. Recent advances in pulmonary embolism diagnosis and management. *Compr Ther* 2001; 27:156-162.
- Hamel E, Pacouret G, Vincentelli D, et al. Thrombolysis or heparin therapy in massive pulmonary embolism with right ventricular dilation: results from a 128-patient monocenter registry. *Chest* 2001; 120:6-8.
- Hamel E, Pacouret G, Vincentelli D, et al. Thrombolysis or heparin therapy in massive pulmonary embolism with right ventricular dilation: results from a 128-patient monocenter registry. *Chest* 2001; 120:120-125.
- Jerges-Sanchez C, Ramierz-Rivera A, Garcia M, et al. Streptokinase and heparin versus heparin alone in massive pulmonary embolism: a randomized control trial. *J Thromb* 1995; 2:227-229.
- Kelly MA, Abbuhl S. Massive pulmonary embolism. Respir Emerg II 1994; 13:547-560.
- Kucher N, Rossi E, De Rosa M, Goldhaber SZ. Massive pulmonary embolism. *Circulation* 2006;113:577-582.
- Kurkciyan I, Meron G, Sterz F, et al. Pulmonary embolism as cause of cardiac arrest: presentation and outcome. *Arch Internal Med* 2000; 160:1529-1535.
- Konstantinides S, Geibel A, Olschewski M, et al. Association between thrombolytic treatment and the prognosis of hemodynamically stable patients with major pulmonary embolism: results of a multicenter registry. Circ J Am Heart Assoc 1997; 96:882-888.
- Lee AY, Hirsh J. Diagnosis and treatment of venous thromboembolism. *Annu Rev Med* 2002; 53:15-33. Piazza G, Goldhaber SZ. Acute pulmonary embolism: part I: epidemiology and diagnosis. *Circulation* 2006;114:e28-e32.
- Piazza G, Goldhaber SZ. Acute pulmonary embolism: part II: treatment and prophylaxis. *Circulation* 2006;114:e42-e47.

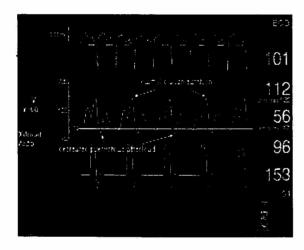
RATIONALE (10)

Answer: D

Typically, the intra-aortic balloon pump (IABP) is inserted percutaneously through the femoral artery. The tip is advanced up the aorta until it is positioned just before the left subclavian artery. Once in position, the balloon is programmed to inflate at the onset of ventricular diastole and deflate just before the beginning of ventricular systole. The balloon is inflated with helium and displaces approximately 40 mL of blood, increasing peak diastolic pressure and improving coronary perfusion and mean arterial pressure. Deflation of the balloon just prior to systole decreases end-diastolic pressure and impedance to the left ventricular ejection. The timing of the IABP is based on the patients ECG. Irregular heart rhythm increases the risk of poor synchronization of the balloon pump and cardiac contraction.

The figure below shows a balloon pump that is timed appropriately with the ECG. The labeled areas of the volume wave form in this figure show the diastolic augmentation during IABP-supported contractions and the decreased presystolic afterload prior to non-augmented-IABP cardiac contractions. The IABP inflation rate to patient's heart rate is 1:2.

Even if the IABP is functioning well, serious complications are not uncommon. The most common complication seen with the IABP is leg ischemia. Even with correct balloon position, aortic perforation, thrombocytopenia, and thromboembolism are other known complications. Reversible renal and hepatic insufficiency can occur with the malposition of the balloon. The balloon can migrate over a branch of the aorta and decrease arterial outflow enough to cause distal hypoperfusion. Most of the time, correctly positioning or taking out the balloon will allow the affected organ to reperfuse and recover.



# **REFERENCES (10)**

Christenson JT, Simonet F, Badel P, et al. Optimal timing of preoperative intraaortic balloon pump support in the high-risk coronary patients. *Ann Thorac Surg* 1999; 68:934-939.

Kumbasar SD, Semiz E, Sancaktar O, et al Mechanical complications of intraaortic balloon counterpulsation. *Int J Cardiol* 1999; 70:69-73.

Robicsek F, Masters TN, Rice H, et al. Enhancing the applicability and effectiveness of intra-aortic balloon counterpulsation. *J Cardiac Surg* 1990; 5:321-327.

Shin H, Yozu R, Sumida T, et al. Acute ischemic hepatic failure resulting from intraaortic balloon pump malposition. *Eur J Cardio-Thorac Surg* 2000; 112:492-494.

RATIONALE (11)

Answer: E

The combination of risk factors, symptoms, and the livedo reticularis make (E) the only correct answer. Cholesterol crystal embolization, also known as atheroembolic disease, is caused by showers of cholesterol crystals from an atherosclerotic aorta that occludes small arteries and arterioles. Although cholesterol crystal embolization can occur spontaneously, it is increasingly recognized as an iatrogenic complication from invasive vascular procedures, such as manipulation of the aorta during angiography or vascular surgery. The use of anticoagulants and fibrinolytics can also precipitate cholesterol embolization. These agents may prevent the formation of a protective thrombus overlying an ulcerated plaque or may initiate the disruption of a complex plaque by intraplaque hemorrhage.

Common embolic sites include the skin, kidney, and pancreas, as well as the gastrointestinal tract and the central nervous system. Cutaneous manifestations include the "purple toe" syndrome and livedo reticularis (blue reticular discoloration of the extremities that produces a lacy, irregular appearance outlining central areas of normal-appearing skin). The cholesterol emboli cause an inflammatory reaction that leads to an endothelial reaction, causing intimal proliferation and intravascular fibrosis of the affected vessels, leading to ischemia or infarction of tissue distal to the emboli.

Cholesterol crystal embolism can also cause renal failure. Presentation may be abrupt. More frequently, a progressive loss of renal function occurs over weeks. Renal outcome varies from complete recovery to dialysis-dependent renal failure.

Laboratory findings include an elevation of the erythrocyte sedimentation rate, eosinophilia, and hypocomplementemia. Most therapeutic interventions are of little benefit once embolization has occurred. Anticoagulation is controversial and may actually contribute to the development of a cholesterol emboli by causing hemorrhage into an atherosclerotic plaque. The benefits of corticosteroids and antiplatelet drugs are questionable. Surgical removal of large emboli has been done with variable success. There is recent evidence that statin-induced plaque stabilization may decrease the extent of embolic induced injury. In general, prognosis is poor.

# **REFERENCES (11)**

- Applebaum RM, Kronzon I. Evaluation and management of cholesterol embolization and the blue toe syndrome. *Curr Opinion Cardiology* 1996; 11:533-542.
- Gaya DR, Foulis AK, Morris AJ. Image of the month. Cholesterol embolization. *Gastroenterology* 2006;130:631, 1022.
- Geraets DR, Hoehns JD, Burke TG, Grover-McKay M. Thrombolytic-associated cholesterol emboli syndrome: case report and literature review. *Pharmacotherapy* 1995; 15:441-450.
- Graziani G, Santostasi S, Angelini C, Badalamenti S. Corticosteroids in cholesterol emboli syndrome. *Nephron* 2001; 87:371-373.
- Hirano Y, Ishikawa K. Cholesterol embolization syndrome: how to recognize and prevent this potentially catastrophic iatrogenic disease. *Intern Med* 2005;44:1209-1210.
- Kazancioglu R, Erkoc R, Bozfakioglu S, et al. Clinical outcomes of renal cholesterol crystal embolization. *J Nephrol* 1999; 12:266-269.
- Pennington M, Yeager J, Skelton H, Smith KJ. Cholesterol embolization syndrome: cutaneous histopathological features and the variable onset of symptoms in patients with different risk factors. *Br J Dermatol* 2002; 146:511-517.

RATIONALE (12)

Answer: B

Milrinone, a second-generation phosphodiesterase inhibitor, is a nonadrenergic inotrope. Inhibiting phosphodiesterase increases intracellular cyclic adenosine monophosphate, resulting in positive inotropy and peripheral vasodilation. The drug may be utilized for decompensated heart failure, as well as for weaning from a cardiopulmonary bypass after cardiac surgery. Milrinone decreases both left ventricular afterload and pulmonary vascular resistance. Cardiac output is increased.

In this patient, the vasodilatory properties of milrinone decreased systemic BP. The cardiac index increased. Pulmonary artery occlusion pressure decreased.

Nitroglycerin is a venous vasodilator that would decrease systemic BP and right atrial pressure, but the cardiac index would not be increased. Dopamine has both inotropic and chronotropic properties. Dopamine would increase the heart rate and cardiac index, but would not lower the BP. Norepinephrine would have inotropic effects, but the  $\alpha$ -adrenergic effects would raise the systemic BP and increase SVR.

Despite the favorable hemodynamic profile for cases of decompensated congestive heart failure (CHF), a recent randomized controlled trial of milrinone (versus placebo) in CHF failed to show a decrease in the number of days hospitalized for cardiovascular causes. There was no significant difference in mortality. The authors concluded that IV milrinone should not be used routinely for cases of decompensated CHF.

#### **REFERENCES (12)**

Cuffe MS, Califf RM, Adams KF, et al. Short-term intravenous milrinone for acute exacerbation of chronic heart failure: a randomized controlled trial. *JAMA* 2002; 287:1541-1547.

Kumar A, Parrillo JE. Shock: classification, pathophysiology, and approach to management. In Critical Care Medicine: Principles of Diagnosis and Management in the Adult. 2nd ed. Parrillo JE, Dellinger RP, eds. St. Louis, MO: Mosby; 2001, 371-420.

RATIONALE (13)

Answer: B

Only one of the possible choices, nesiritide, is consistent with the hemodynamic and diuretic properties in the clinical scenario. Nesiritide is recombinant human brain natriuretic peptide. Similar to the endogenous hormone that is released with increased myocardial wall tension from volume overload, nesiritide has vasodilatory effects (both arterial and venous), increasing cardiac output without direct inotropic or chronotropic effects. Nesiritide also has a direct diuretic effect, increasing glomerular filtration rate and sodium excretion.

Dopamine may increase cardiac output, but the heart rate would also be increased. Milrinone is a phosphodiesterase III inhibitor that has vasodilating propeties. Milrinone would decrease filling pressures and increase cardiac output but would not have such a potent diuretic effect. Nitroglycerin would decrease filling pressures but would not increase cardiac output or have a potent diuretic effect. Bumetanide is a loop diuretic and cardiac output would not be increased.

In a recent trial of nesiritide versus nitroglycerin for treatment of decompensated heart failure, nesiritide improved hemodynamic function (lowered pulmonary capillary occlusion pressure) more effectively than nitroglycerin. However, there was no difference in subjective dyspnea, and relief of other clinical symptoms was only modestly better with nesiritide.

#### **REFERENCES (13)**

- Marcus LS, Hart D, Packer M, et al. Hemodynamic and renal excretory effects of human brain natriuretic peptide infusion in patients with congestive heart failure. A double-blind, placebo-controlled, randomized crossover trial. *Circ J Am H Assoc* 1996; 94(12):3184-3189.
- Publication Committee for the VMAC Investigators. Intravenous nesiritide vs. nitroglycerin for treatment of decompensated heart failure. *JAMA* 2002; 287:1531-1540.
- Sackner-Bernstein J, Aaronson KD. Nesiritide for acute decompensated heart failure: does the benefit justify the risk? *Curr Cardio Rep* 2007;9(3):187-193.
- Shah SJ, Teerlink JR. Nesiritide: A reappraisal of efficacy and safety. Expert Opin Pharmacother 2007;8(3):361-369.
- van der Starre PJ. Nesiritide in cardiovascular anesthesia. Curr Opin Anaesthesiol 2005;18(1):83-87.

RATIONALE (14)

Answer: C

The intraaortic balloon pump (IABP) is an important and established therapy for various serious cardiac conditions. The underlying principle of the IABP is that inflating the balloon during early diastole will displace blood in the aorta, increasing the diastolic BP, and by this mechanism improve coronary perfusion pressure; deflation of the balloon just before systole will decrease left ventricle afterload by producing a rapid decrease in aortic pressure. This, in turn, will decrease myocardial oxygen demand and consumption. Hemodynamic effects that would be expected in this clinical scenario include the following:

- Increase in the diastolic arterial pressure, systolic arterial pressure, and mean arterial pressure;
- Decrease of the pulmonary artery occlusion pressure;
- Improved left ventricular performance and ejection fraction, with increased cardiac putput and cardiac input; and
- Decreased myocardial oxygen demand that would result in decreased oxygen consumption.

Only the units in answer C match all these changes, making it the most correct answer.

# **REFERENCES (14)**

Goldstein DJ, Oz MC, Rose EA. Implantable left ventricular assist devices. N Engl J Med 1998; 339:1522.

Kelly R. Intra-aortic Balloon Counterpulsation. In Critical Care Medicine. Parrillo JE, Dellinger RP, eds. St. Louis, MO: 2001; 91-106.

RATIONALE (15)

Answer: D

This rhythm strip represents a polymorphic ventricular tachycardia also known as a Torsades de Pointes. Torsades is associated with prolongotion of the QT interval. Many drugs utilized in the ICU can prolong the QT interval and result in a Torsades. Some commonly cited culprits include quinidine, sotalol, terfenadine, astemizole, cisapride, erythromycin IV, haloperidol, pentamidine, procainamide, ibutilide, trimethoprim-sulfa, and tricyclic antidepressants.

The treatment for Torsades includes prompt discontinuation of the offending medication and an IV of magnesium sulfate 2 g, with repeated doses at 10 to 15 minute intervals, as required. Other electrolyte abnormalities should be corrected as well.

Utilization of traditional antiarrhythmic drugs such as amiodarone, quinidine, and verapamil are ineffective in terminating this arrhythmia, and can potentially worsen the clinical situation. Calcium carbonate and insulin+ glucose is utilized in treating ECG changes produced by hyperkalemia and has no effect on Torsades.

# **REFERENCES (15)**

Chaudhry GM, Haffajee CI. Antiarrhythmic agents and proarrhythmia. Crit Care Med 2000;28(suppl): N158-N164.

Francis GS. Cardiac complications in the intensive care unit. Clin Chest Med 1999; 20:269-286.

Gupta A, Lawrence AT, Krishnan K, Kavinsky CJ, Trohman RG. Current concepts in the mechanisms and management of drug-induced QT prolongation and torsade de pointes. Am Heart J 2007;153:891-899.

Kannankeril PJ, Roden DM. Drug-induced long QT and torsade de pointes: Recent advances. *Curr Opin Cardiol* 2007;22:39-43.

Tzivoni D, Keren A, Cohen AM. Treatment of Torsade de Pointes with magnesium sulfate. Circ J Am Heart Assoc 1988; 77:392-396.

RATIONALE (16) Answer: B

Evaluation of the volume and contour of the arterial pulse waveform can provide significant clues as to the etiology of a wide variety of cardiovascular abnormalities. The bisferious pulse characterized by two systolic peaks occurs during conditions in which a large stroke volume is rapidly ejected from the left ventricle. In patients who have pure aortic stenosis, the second wave tends to be higher than the first, and the pulse rises slowly.

The arterial waveform in patients who have dilated cardiomyopathy and other low-output states is notable for two pulsations, the second of which occurs in diastole. The second wave is felt to be the result of a reduction in the normal pressure pulse, in association with the usual incisural downslope and rebound.

The immigration of large numbers of individuals from other countries to the United States has led to the resurgence of numerous clinical problems which had decreased in severity over the previous decades. Tuberculosis, with its many and varied complications, is endemic in many foreign countries. The jugular venous pulse accompanying tuberculosis-induced constrictive pericarditis is notable for the prominent x- and y-descents. The y-descent tends be deeper and more rapid than the x-descent and may be accompanied by a loud third heart sound, the so-called "pericardial knock." This sound is the result of the abrupt dessation of diastolic falling due to the restrictive effects of the diseased pericardium.

# **REFERENCES (16)**

Heart disease. *In*: Braunwald's heart disease: a textbook of cardiovascular medicine, single volume. Braunwauld E, ed. Philadelphia, PA: WB Saunders; 1988.

Ewer M, Naccarelli G. Cardiac critical care. Crit Care Clin 1989; 5:3.

RATIONALE (17) Answer: D

This patient has developed an arrhythmia as a consequence of severe hyperkalemia. Even though this patient had a history of renal disease, her potassium levels were not checked when she presented in the emergency room, and she was intubated. As part of that process, she received succinylcholine, which can transiently increase serum levels of potassium, leading to this arrhythmia.

Serum levels of potassium in this patient were 8.6 mEq/L at the time the arrhythmia occurred. Hyperkalemia has multiple effects on cardiac rhythm, and the typical changes seen with hyperkalemia that are present in an ECG include: peaked T-waves, loss of P-waves, widening QRS complexes, sine wave, ventricular arrhythmias, and asystole. As patients develop increasing levels of serum potassium, these ECG changes progressively develop and can lead to the life-threatening arrhythmias, as seen in the figure.

The ECG below depicts loss of P-waves, peaked T-waves, and a significant widening of the QRS complexes. The treatment of hyperkalemia is aimed at stabilizing the myocardial tissue, and for that calcium chloride or calcium gluconate are the drugs of choice. Transient movement of extracellular serum potassium into cells can be achieved with the administration of insulin 10 units IV plus glucose to prevent hypoglycemia, and in some cases, administration of sodium bicarbonate. A  $\beta$ -agonist inhaler can also be used for this. More definitive removal of potassium from the body is achieved with resin binding enemas (sodium polystyrene), and with hemodialysis. The patient was given calcium chloride and was emergently dialyzed, with resolution of the ECG changes and arrhythmias.

# **REFERENCES (17)**

Gennari FJ. Disorders of potassium homeostasis. Hypokalemia and hyperkalemia. Crit Care Clin 2002; 18:273-288.

Mazze RI, Szescue HM, Houston JB. Hyperkalemia and cardiovascular collapse following administration of succinylcholine to the traumatized patient. *Anesthesiology* 1969; 31:540-547. Zanotti S, Dellinger RP. Hyperkalemia and hypokalemia. *In* Textbook of critical care medicine. 5th ed. Fink M, Abraham E, Kochanek P, Vincent JL, eds. Philadelphia, PA: Saunders; 2005.

RATIONALE (18)

Answer: C

This patient is having an acute S-T elevation anterior myocardial infarction. In addition, the patient presents signs of developing cardiogenic shock with tachycardia, low BP, and signs of a cardiogenic pulmonary edema. The SHOCK randomized trial demonstrated that in patients with acute myocardial infarction, complicated by cardiogenic shock, early mechanical revascularization reduced mortality, as compared with initial medical stabilization followed by late or no revascularization. Based on these findings, in their most recent guidelines the American College of Cardiology and the American Heart Association elevated early mechanical revascularization for cardiogenic shock to a class I recommendation for patients younger than 75 years with a S-T elevation or a left-bundle branch block acute myocardial infarction.

Although all the other options include treatments that are important for patients with acute myocardial infarction, some of them may be deleterious in patients developing cardiogenic shock, and may have negative impacts on patients with hypertension. In hospitals without the capability for onsite cardiac interventions, stabilization and transfers to an institution capable of performing revascularization is probably the best strategy with acute myocardial infarction and cardiogenic shock.

The DANAMI trial compared primary mechanical revascularization (angioplasty) with thrombolytic therapy in patients with S-T elevation acute myocardial infarction. Patients randomized at sites without cardiac catheterization facilities were transferred for revascularization. In this trial, the combined outcome of death, recurrent myocardial infarction, and stroke was improved in patients randomized to the mechanical revascularization arm (whether it was done onsite or the patient was transferred to another facility).

# **REFERENCES (18)**

Anderson HR, Nielson TP, Rasmussen K, et al. A comparison of coronary angioplasty with fibrinolytic therapy in acute myocardial infarction. N Engl J Med 2003; 349:733-742.
Babaev A, Frederick PD, Pasta DJ, et al. Trends in management and outcomes of patients with acute myocardial infarction complicated by cardiogenic shock. JAMA 2005; 294:448-454.
Hochman JS, Sleeper LA, Webb JG, et al. SHOCK Investigators. Early revascularization and acute myocardial infarction complicated by cardiogenic shock. N Engl J Med 1999; 341:625-634.

RATIONALE (19)

Answer: B

This patient is presenting with an acute myocardial infarct with S-T elevations in the inferior leads. The development of hypotension after administration of nitroglycerin, in addition to his clinical picture of increased jugular venous distension and clear lungs, is consistent with a right ventricular infarction. Right ventricular infarction occurs in up to 50% of inferior left ventricular infarctions.

It is important to recognize this potential complication, since it may effect choice of treatment for these patients. The diagnosis of right ventricular infarction can sometimes be confirmed by the performance of an ECG with right precordial chest leads. Patients with right ventricular infarctions respond very poorly to decreases in preload like those caused by nitroglycerin; they also could respond poorly to decreases in right ventricular inotropy for  $\beta$ -blockers and will be unable to tolerate arrhythmias, such as atrial fibrillation.

The use of a pulmonary artery catheter can help differentiate this complication, secondary to typical changes that are seen in a right ventricular infarction on the hemodynamic profile. Of the profiles presented in the question, profile B is the most consistent with the expected findings in a patient who is hypotensive from right ventricular infarction. Usually right atrial pressures are affected by an increased centralvenous pressure; there are also increases in the pulmonary artery pressures, the wedge pressure or the pulmonary artery occlusion pressure will usually be normal or decreased, and, because of decreased filling of the left ventricular, the cardiac index will be decreased. The other profiles are not consistent with this pattern and, therefore, will be less likely to be found in a patient with right ventricular infarction.

# **REFERENCES (19)**

Haji SA, Movahed A. Right ventricular infarction: diagnosis and treatment. *Clin Cardiol* 2000; 23:473-482.

Jacobs AK, Leopold JA, Bates E: Cardiogenic shock caused by right ventricular infarction. *J Am Coll Cardiol* 2003; 1:1273-1279.

Kinch JW, Ryan TJ. Right ventricular infarction. N Engl J Med 1994; 330:1211-1217.

Manoharan G, De Bruyne B. Right ventricular myocardial infarction. Heart 2005;91:e40.

Moye S, Carney MF, Holstege C, et al. The electrocardiogram in right ventricular myocardial infarction. Am J Emerg Med 2005;23:793-799.

O'Rourke RA, Dell'Italia LJ. Diagnosis and management of right ventricular myocardial infarction. Curr Probl Cardiol 2004;29:6-47.

RATIONALE (20)

Answer: E

Hemodynamic support is the cornerstone of the management of patients with severe sepsis and septic shock. The shock profile associated with sepsis is complex and involves several abnormalities in the cardiovascular system. Abnormalities associated with severe sepsis and septic shock include the following:

- Intravascular volume depletion secondary to increased capillary leakage with inflammation;
- Hypotension with profound vasodilation; and
- Decreased cardiac contractility, which in the early phases of sepsis is associated with a decreased cardiac output.

In the early phases of sepsis, hemodynamic support is aimed at correcting these abnormalities. Goals of hemodynamic support in the first 6 hours include:

- Correction of intravascular volume as measured by a central venous pressure (CVP) of 8-12 mm Hg;
- Maintenance of adequate tissue perfusion as measured by a mean arterial pressure (MAP) ≥65 mm Hg; and
- Maintenance of adequate cardiac output as measured by a Scvo<sub>2</sub>>70%.

In the case given in the question, there are several hemodynamic endpoints that have not been met. The CVP is 6 mm Hg, MAP is 64 mm Hg, and the Scvo<sub>2</sub> is 60%. The first goal must be restoration of intravascular volume, as measured by a CVP of 8-12 mm Hg. The correct answer at this point is to give more fluids.

# **REFERENCES (20)**

Dellinger RP, Carlet JM, Masure H, et al. Surviving Sepsis Campaign Management Guidelines Committee: Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med* 2004; 32:858-873.

Hollenberg SM, Ahrens TS, Annane D, et al. Practice parameters for the hemodynamic support of sepsis in adult patients: 2004 update. *Crit Care Med* 2004; 32:1928-1948.

RATIONALE (21)

Answer: C

This patient is in anaphylactic shock, most likely as a reaction to the contrast material used in the CT scan. An anaphylactic shock is the most severe presentation of an anaphylactics. Anaphylactics can be triggered by immunologic or nonimmunologic mechanisms. Agents associated with anaphylactoid reactions include radiocontrast dye, opiates, aspirin, and other nonsteroidal antiinflammatory drugs. The cornerstone of therapy for an anaphylactic shock includes prompt administration of IV fluids and epinephrine. Epinephrine can be administered subcutaneous, intramuscularly, or intravenously. In patients with clear signs of hypoperfusion and shock, subcutaneous administration is not recommended. For treatment of anaphylactic shock, epinephrine should be administered intramuscularly or intravenously. IV administration is reserved for cases of severe hemodynamic collapse; extreme caution is necessary in patients at risk for myocardial ischemia.

Corticosteroids have a role in the later treatment of anaphylactics, mostly to prevent late phase reactions such as intermediate airway edema and bronchoconstriction. However, corticosteroids have no immediate effect on hemodynamic abnormalities in anaphylactic shock. Dobutamine is an inotrope and will not help improve the patient's BP. Although Dopamine is a catecholamine with vasopressor activity, because of its lack of effect on other processes in anaphylaxis, it would not be the first choice.

# **REFERENCES (21)**

Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. part 8: Advanced challenges in resuscitation: section 3: special challenges in ECC. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. *Circ J Am Heart Assoc* 2000; 1229-1252.

Montanaro A, Bardana EJ Jr. The mechanisms, causes, and treatment of anaphylaxis. *J Investig Allergol Clin Immunol* 2002; 12:2-11.

Silverman HJ, Van Hook C, Haponik EF. Hemodynamic changes in human anaphylaxis. *Am J Med* 1984; 77:341-344.

RATIONALE (22)

Answer: C

The patient in this question is a young male who presents with complains of acute onset of pleuritic chest pain and shortness of breath. The ECG shown in the figure is significant for PR depression and diffuse ST-segment elevations in the precordial leads. The clinical picture, in conjunction with the ECG findings, are most suggestive of acute pericarditis. Acute pericarditis can occur in a very broad list of diseases, both common and exotic. Most patients with acute pericarditis will present with complaints of chest pain, mostly central and usually pleuritic. The ECG is a valuable adjunct in making the diagnosis of acute pericarditis. Commonly found ECG abnormalities include: ST-segment elevations (J point) diffusely spread in several leads, usually with PR segment depressions in most leads, and the reverse always in a VR, usually V1.

Additional findings that support a diagnosis of acute pericarditis include a pericardial rub. Regarding this question, the most appropriate next step would be a transthoracic echocardiogram of the heart to evaluate possible associated pericardial effusion and any hemodynamic compromise from it.

At this point, a cardiac catherization is not indicated, as there is no evidence of active myocardial ischemia. Based on the available clinical data, a CT scan of the chest with an IV contrast and a lower extremity Doppler to rule out thrombolysis is not indicated.

# **REFERENCES (22)**

Spodick DH. Acute pericarditis. JAMA. 2003; 289:1150-1153.

Spodick DH. Diagnostic electrocardiographic sequences in acute pericarditis: significance of PR segment and PR vector changes. Circ J Am Heart Assoc 1973: 48:575-580.

RATIONALE (23)

Answer: B

The patient in this question presents with a constellation of symptoms that include hypotension, bradycardia, and altered mental status. This presentation is consistent with either a calcium channel blocker or  $\beta$ -blocker toxicity. In this case, the patient had a calcium channel blocker toxicity secondary to verapamil.

Calcium channel blocker toxic syndromes are the most common cause of cardiac-drug-toxicity-related mortality. Calcium channel blockers block the slow calcium channels, decreasing the influx of calcium into the myocardial cells, resulting in decreasing function in the cardiac pacemaker's cells, myocardial cells and arterial smooth muscles. Calcium channel blocker toxicity often presents with significant hypotension and bradycardia. The initial therapy should include gastrointestinal decontamination as in toxic exposures to other medications.

Calcium is the first line treatment for the hemodynamic alterations seen with both  $\beta$ -blockers and calcium channel blockers toxicities. Calcium has been shown to be effective in both animal models and human cases. Administration of calcium increases the extracellular calcium concentrations, allowing

calcium entry via unblocked calcium channels during depolarization. Calcium will improve HR, contractility, and BP. Calcium chloride is preferred over calcium gluconate, as it contains 3 times the amount of elemental calcium on a weight-to-weight basis.

Calcium is best administered via a central venous catheter, if possible. Current recommendations for dosing consists of an initial bolus administration of 10-20 mL of 10% calcium chloride or 30-60 mL of 10% calcium gluconate. Additional therapies for treating calcium channel blocker toxicity include glucagon, catecholamines, and phosphodiesterase inhibitors.

Atropine is usually not beneficial in these patients, as it will not correct the BP. Transcutaneous pacemakers may be necessary in refractory cases, but initial therapy should consist of calcium chloride as mentioned above. Hemodialysis is not indicated for calcium channel blocker toxicity. Finally, an IV pacemaker should be used only as a last resort for refractory bradycardia.

#### **REFERENCES (23)**

DeWitt C, Waksman J. Pharmacology, pathophysiology and management of Calcium Channel Blocker and ß-Blocker Toxicity. *Toxicol Re.* 2004; 23:223-238.

Salhanick SD, Shannon MW. Management of calcium channel antagonist overdose. *Drug Saf* 2003; 26:5-79.

Brent J. Calcium channel-blocking agents. In: Critical care toxicology. Brent J, et al. eds. Philadelphia, PA: Mosby; 2005.

RATIONALE (24)

Answer: A

It has long been recognized that positive airway pressure in lung inflation can have distinct effects on heart loading conditions and performance. Other direct and indirect effects on the heart have been suspected as well. Sustained increased intrathoracic pressure produces a net decrease in venous return and a decrease in stoke volume and cardiac output. This clinical effect needs to be differentiated from the dynamic immediate effects of intrathoracic pressure variations. Lung inflation with positive airway pressure may have dynamic effects on myocardial contractile status that can have rapid onset, even during a single breath.

Positive-pressure inspiration, inferior cava constriction, and release of abdominal compression have all been reported to decrease right ventricular (RV) inflow. RV inflow is increased with positive-pressure expiration, the release of inspiratory hold, the constriction of the inferior vena cava, and abdominal compression. Decrease of the RV end flow during positive-pressure inspiration and vena cava constriction transiently decreases RV end diastolic volume and increases the transeptal pressure gradient, causing the septum to shift to the right.

With left ventricular (LV) end diastolic volume, the result is no change in the anterior/posterior diameter and a stroke volume increase; when RV inflow increases, the opposite occurs. Therefore, during the expiratory phase of mechanical ventilation, and when the inspiratory hole and the cable constriction are released, or during abdominal compression, the transseptal pressure gradient decreases, causing the septum to shift to the left. LV end diastolic pressure and stroke volume decrease as a result.

Mechanical ventilation can affect cardiac function through a number of mechanisms. Direct ventricular interaction (change in the volume of one ventricle causing a simultaneous and opposite change in the volume of the other ventricle) is one of the mechanisms by which positive end expiratory pressure may decrease the left ventricular end diastolic volume in output during mechanical ventilation. These affects are dependant on the presence of pericardial constraint. A sudden decrease in right ventricular end diastolic volume is associated with a simultaneous increase in left ventricular end diastolic volume, whereas a sudden increase in right ventricular end diastolic volume is associated with a decrease in left ventricular end diastolic volume.

#### **REFERENCES (24)**

- Brower R, Wise RA, Hassapoyannes C, Bromberg-Barnea B, Permutt S. Effect of lung inflation on lung blood volume and pulmonary venous flow. *J Appl Physiol* 1985;58:954-963.
- Cassidy SS, Eschenbacher WL, Robertson CH Jr, Nixon JV, Blomqvist G, and Johnson RL Jr.
- Cardiovascular effects of positive-pressure ventilation in normal subjects. *J Appl Physiol* 1979;47:453-461.
- Cassidy SS, Mitchell JH, Johnson RL Jr. Dimensional analysis of right and left ventricles during positive-pressure ventilation in dogs. *Am J Physiol Heart Circ Physiol* 1982;242:H549-H556.
- Fessler HE, Brower RG, Wise RA, Permutt S. Effects of positive end-expiratory pressure on the gradient for venous return. *Am Rev Respir Dis* 1991;143:19-24.
- Fewell JE, Abendschein DR, Carlson CJ, Murray JF, Rapaport E. Continuous positive-pressure ventilation decreases right and left ventricular end-diastolic volumes in the dog. *Circ Res* 1980; 46:125-132.
- Haney MF, Johansson G, Haggmark S, Biber B. Myocardial systolic function increases during positive pressure lung inflation. *Anesth Analg* 2005;101:1269-1274.
- Luecke T, Pelosi P. Clinical review: Positive end-expiratory pressure and cardiac output. Crit Care Med 2005;9:607-621.
- Mitchell JR, Whitelaw WA, Sas R, Smith ER, Tyberg JV, Belenkie I. RV filling modulates LV function by direct ventricular interaction during mechanical ventilation. *Am J Physiol Heart Circ Physiol* 2005;289:H549-H557.
- Rankin JS, Olsen CO, Arentzen CE, et al. The effects of airway pressure on cardiac function in intact dogs and man. *Circulation* 1982; 66:108-120.

RATIONALE (25)

Answer: D

Circulating levels of vasopressin are elevated during hypovolemic, cardiogenic and obstructive shock. However, such a sustained response does not occur in septic shock. Initially, vasopressin levels are significantly elevated; over the ensuing days of sepsis/septic shock, these levels fall, approaching normal levels between day 2 and 3. With increasing doses of vasopressin, blood flow is reduced to the skin, skeletal muscle, bowel and fat.

Vasopressin induces an increase in mean arterial pressure and a decrease in cardiac output, mainly linked to negative chromotropic effect. Rebound hypotension often occurs when the drug is discontinued. Vasopressin can significantly decrease platelet count during infusion. Vasopressin at doses of 0.04 or less have minimal effect on the cardiac index. Normal individuals have no blood pressure effects during exogenous administration of vasopressin up to levels of 0.26 U/min. However, for patients in shock, a hypersensitivity to vasopressin has been noted, with a confidant decrease in the effectiveness of the catecholamines.

#### **REFERENCES (25)**

- Barrett LK, Singer M, Clapp LH. Vasopressin: Mechanisms of action on the vasculature in health and in septic shock. *Crit Care Med* 2007;35:33-40.
- Bassi G, Radermacher P, Calzia E. Catecholamines and vasopressin during critical illness. Endocrinol Metab Clin North Am 2006;35:839-857, x.
- Dunser MW, Mayr AJ, Ulmer H, et al. The effects of vasopressin on systemic hemodynamics in catecholamine-resistant septic and postcardiotomy shock: a retrospective analysis. *Anesth Analg* 2001;93:7-13.
- Holmes CL, Patel BM, Russell JA, Walley KR. Physiology of vasopressin relevant to management of septic shock. *Chest* 2001;120:989-1002.
- Holmes CL, Walley KR. Vasopressin in the ICU. Curr Opin Crit Care 2004;10:442-448.
- Kaplan NM, Palmer BF, Chen P. Vasopressin: new uses in critical care. Am J Med Sci 2002;324:146-154.
- Manoharan G, De Bruyne B. Right ventricular myocardial infarction. *Heart* 2005;91:e40.
- Moye S, Carney MF, Holstege C, et al. The electrocardiogram in right ventricular myocardial infarction. *Am J Emerg Med* 2005;23:793-799.
- O'Rourke RA, Dell'Italia LJ. Diagnosis and management of right ventricular myocardial infarction. Curr Probl Cardiol 2004;29:6-47.metabolic effects of low-dose vasopressin infusions in vasodilatory septic shock. Crit Care Med 2001;29:487-493.
- Strohmenger HU, Krismer A, Wenzel V. Vasoressin in shock states. *Curr Opion Anaesthesiol* 2003;15:159-164.
- Tsuneyoshi I, Yamada H, Kakihana Y, Nakamura M, Nakano Y, Boyle WA. Hemodynamic and metabolic effects of low-dose vasopressin infusions in vasodilatory esptic shock. *Crit Care Med* 2001:29:487-493.

RATIONALE (26)

Answer: D

This patient's pulmonary artery hypertension is likely due to a pulmonary embolism. He is requiring vasopressor levels of dopamine and has a central venous pressure (CVP) of 12 mm Hg. It is generally accepted that fluid resuscitation effects are variable in patients with suspected pulmonary embolism, depending upon the degree of pulmonary artery hypertension and current intravascular volume status.

A CVP of 12, however, would be considered inadequate, with CVPs of 20-25 in the optimal range. Thrombolytic therapy may on occasion be utilized in patients who are high risk for pulmonary embolisms, but have only echocardiographic support for pulmonary embolism. In this patient, a definitive test would be optimal management, as he is adequately oxygenated with supplemental  $O_{2}$ ; requires levels of dopamine that would not be considered high; and has a relative contraindication to thrombolytic therapy.

Since a CT scan of the chest is the only test currently available, the choice is between dye exposure, which in a patient with chronic renal insufficiency carries a significant risk of worsening that condition, versus thrombolytic exposure, with its risk of intrarenal hemorrhage. The risks associated with the use of the dye in a patient with renal insufficiency, even if it results in the need for renal replacement therapy, when weighed against the risk of an intracranial hemorrhage, gives the edge to immediate definitive testing for the presence or absence of a pulmonary embolism. Delaying testing in this patient may allow his condition to deteriorate. Given these considerations, urgent thrombolytic therapy would not be the most appropriate choice.

#### **REFERENCES (26)**

- Carbone R, Bossone E, Bottino G, et al. Secondary pulmonary hypertension: diagnosis and management. Eur Rev Med Pharmacol Sci 2005;9:331-342.
- Dong B, Kirong Y, Liu G, et al. Thrombolytic therapy for pulmonary embolism. *Cochrane Database Syst Rev* 2006;2:CD004437.
- Goldhaber SZ. Thrombolysis in pulmonary embolism: a debatable indication. *Thromb Haemost* 2001;86:444-451.
- Jardin F, Vieillard-Baron A. Monitoring of right-sided heart function. *Curr Opin Crit Care* 2005;11:271-279.
- Meyer FJ, Schoene AM, Borst MM. Pathophysiological aspects of cardiopulmonary interaction. *Clin Nephrol* 2003;60(suppl 1):S75-S80.
- Perrier A, Roy PM, Sanchez O, et al. Multidector-row computed tomography in suspected pulmonary embolism. *N Engl J Med* 2005;352:1760-1768.
- Segal JB, Streiff MB, Hoffman LV, et al. Management of venous thromboembolism; a systematic review for a practice guideline. *Ann Intern Med* 2007;146:211-222.
- Via G, Braschi A. Pathophysiology of severe pulmonary hypertension in the critically ill patient. Min *Anestesiol* 2004;70:233-237.

RATIONALE (27)

Answer: E

Effusive-constrictive pericarditis is a pericardial syndrome characterized by tense pericardial effusion with a tamponade, in addition to a visceral pericardium constriction. Diagnostic criterion is the failure of the right atrial pressure to fall by 50% or more, or to a level below 10 mm Hg after intra-pericardial pressure is normalized. Although the most frequent cause is idiopathic, followed by neoplastic, a higher percentage of patients with radiation-induced pericarditis will also have an effusive-constrictive pericarditis. Following the drainage of the pericardial effusion, there is typically some improvement in the cardiac index, although it may not be as dramatic as it was in this case. In some cases of constrictive

pericarditis associated with idiopathic acute exudative pericarditis, spontaneous resolution is possible. This is also called transient cardiac constriction. In other cases, surgery may be required to relieve visceral constriction. Prior to pericardiocentesis, the right atrial transmural pressures are very low (less than 2 mm Hg), with a pulsus paradoxus of 10 mm Hg or more. These are typical criteria for a cardiac tamponade and typically resolve with the drainage of the pericardial effusion. However, constriction and elevated right atrial pressure remain. Radiotherapy is a likely risk factor for this patient given her medical history of lung cancer not in remission and the fact she received previous therapy.

#### **REFERENCES (27)**

Sagrista-Sauleda J, Angel J, Sanchez A, et al. Effusive-constrictive pericarditis. *N Engl J Med* 2004;350:469-475.

Ling LH, Oh JK, Schaff HV, et al. Constrictive pericarditis in the modern era: evolving clinical spectrum and impact on outcome after pericardiectomy. *Circulation* 1999;100:1380-1386.

Sagrista-Sauleda J, Permanyer-Miralda G, Candell-Riera J, et al: Transient cardiac constriction: an unrecognized pattern of evolution in effusive acute idiopathic pericarditis. *Am J Cardiol* 1987;59:961-966.

RATIONALE (28)

Answer: C

Therapy for hypertrophic cardiomyopathy is directed at the dynamic left ventricular (LV) outflow tract obstruction. The obstruction causes an increase in LV systolic pressure which leads to a complex interplay of abnormalities that decrease cardiac output. In the intensive care setting, this condition often deteriorates with volume depletion, and with the institution of inotropic agents. In that circumstance, the infusion of fluids and the discontinuation of inotropic agents is the initial therapy. A beta blocker should also be added; however, if hypotension is present, a vasoconstrictor such as phenylephrine should be administered first. Acute onset of atrial fibrillation may result in severe hemodynamic compromise due to the loss of atrial contractions. Prompt cardioversion should occur in this circumstance.

The first line approach to the relief of symptoms is to block the effects of catecholamines that exacerbate the outflow tract obstruction, and to slow that heart rate to enhance diastolic feeling. Beta blockers are generally the initial choice to accomplish these goals. Verapamil, the calcium channel blocker, can also be used.

Sudden death has been reported in patients with severe pulmonary hypertension and severe outflow obstruction who are given verapamil. This drug should be given with caution in patients with this combination of findings. Nitroglycerin would decrease cardiac filling and is problematic.

#### **REFERENCES (28)**

- Braunwald E, Seidman CE, Sigwart U. Contemporary evaluation and management of hypertrophic cardiomyopathy. *Circulation* 2002; 106:1312-1316.
- Maron, BJ. Hypertrophic cardiomyopathy: a systematic review. JAMA 2002; 287:1308-1320.
- Nishumura, RA. Hypertrophic obstructive cardiomyopathy. N Engl J Med 2004; 350:1320-1327.
- Semsarian C. Guidelines for the diagnosis and management of hypertrophic cardiomyopathy. *Heart Lung Circ* 2007;16:16-18.
- Sherrid MV. Pathophysiology and treatment of hypertrophic cardiomyopathy. *Prog Cardiovas Dis* 2006;49:123-151.
- Spirito P, Seidman CE, McKenna WJ, Maron BJ. The management of hypertrophic cardiomyopathy. N Engl J Med 1997;336:775-785.

RATIONALE (29)

Answer: E

This patient with severe cardiomyopathy has suffered a ventricular fibrillation cardiac arrest. In addition to antiarrhythmic therapies and insertion of an implantable defibrillator, neuroprotection after the return of spontaneous circulation is essential, to help ensure the best possible neurological outcome for the patient after his cardiac arrest. Therapeutic hypothermia has been shown in two large randomized controlled trials to improve neurologic outcome. Currently, therapeutic hypothermia is recommended by the International Liaison Council on Resuscitation after cardiac arrest.

Achieving therapeutic hypothermia to a target of 33°C (91.4°F) can be challenging. Although numerous novel devices have been developed (and are in development) for the optimal cooling of patients, a strategy has also been described utilizing therapies and devices that are commonly available in routine practice. One of these strategies is to apply cooling blankets and cold IV fluids to achieve a target temperature of 33°C (91.4°F). This is after mechanical ventilation, deep sedation, and neuromuscular blockade have been achieved. Most sources recommend therapeutic hypothermia for 12-24 hours postarrest, with a slow return to a normal temperature of approximately 0.25°C per hour.

#### **REFERENCES (29)**

- Bernard S, Gray T, Buist D, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia, *N Engl J Med* 2002;346;557-563.
- Ewy GA. Cardiocerebral resuscitation: The new cardiopulmonary resuscitation. *Circulation* 2005;111:2134-2142.
- The Hypothermia After Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve neurologic outcome after cardiac arrest. *N Engl J Med* 2002;346:549-556.
- Nolan JP, Morley PT, Vanden Hoek TL, Hickey RW. Therapeutic hypothermia after cardiac arrest, ILCOR Advisory Statement, Circ J Am Heart Assoc 2003;108:118-121.

RATIONALE (30)

Answer: D

There are a number of important physiologic (and specifically hemodynamic) effects of pregnancy that the critical care clinician should be aware of. These would be especially important if a pregnant patient were to become hemodynamically unstable and need cardiovascular support. Pregnancy is associated with an increase in total circulating blood volume (as much as 1,500 mL). However, patients with preeclampsia may not have this typical degree of increased circulating blood volume and, therefore, may be at higher risks for hemodynamic effects with blood loss associated with delivery.

The gravid uterus can decrease venous return to the heart when the patient is in the supine position. Therefore, for a pregnant patient with signs of acute circulatory insufficiency, it is important to place the patient in the left lateral decubitus position to facilitate venous return to the heart.

Despite an increase in total circulating blood volume, there is no demonstrable increase in pulmonary capillary occlusion pressure. The HR is typically mildly elevated. The normal physiology of pregnancy will exhibit a decrease in systemic vascular resistance.

#### **REFERENCE (30)**

Yeomans ER, Gilstrap LC III. Physiologic changes in pregnancy and their impact on critical care. *Crit Care Med* 2005;33(10 suppl):S256-S258.

RATIONALE (31)

Answer: A

After exposure to extracorporeal circulation, an intense proinflammatory response, induced by the cardiopulmonary bypass circuit, can cause profound hemodynamic derangements in the immediate postoperative period from cardiac surgery. This may be further complicated by the presence of myocardial depression, which may be de novo or an exacerbation of preexisting poor myocardial function. The proinflammatory postcardiopulmonary bypass state can be associated with potent peripheral vasodilation.

Use of protamine sulfate has been associated with both immune-mediated as well as idiosyncratic reactions that can manifest as severe hypotension, especially in diabetics. Dynamic changes in myocardial compliance, especially in the setting of hypothermia, can make estimations of cardiac preload based on filling pressures quite challenging. Changes in cardiac output with intravascular volume expansion may be better at identifying preload dependency as compared to pressure-based assessments.

If the patient developed a pulseless electrical activity, cardiac tamponade could be the etiology. However, there would be no time to bring the patient back to the operating room, and emergent bedside resternotomy in the ICU (in the hands of qualified personnel) would be indicated to evacuate any clot that could be obstructing cardiac output.

A high urine output is not necessarily a good indicator of volume status, as a post-cardiopulmonary bypass diuresis is common immediately post-cardiac surgery.

#### **REFERENCE (31)**

St Andre AC, DelRossi A. Hemodynamic management of patients in the first 24 hours after cardiac surgery. *Crit Care Med* 2005;33:2082-2093.

RATIONALE (32)

Answer: C

Concerns over postoperative bleeding are common in the ICU after cardiac surgery. Most of the bleeding is considered a medical (related to platelet dysfunction or other acquired coagulopathies) rather than a surgical (technical procedure-related) problem of hemostasis.

Dysfunction of platelets is common, and is usually the result of hypothermia or exposure to the cardiopulmonary bypass circuit. As systemic heparin administration is required for cardiopulmonary bypass, a patient may need additional dosing of protamine to correct any acquired coagulopathy associated with heparin.

Thrombocytopenia is also common and often warrants platelet transfusion in the ICU.

The decision to take a patient back to the operating for reexploration after cardiac surgery is a function of the total output of blood from the mediastinal tubes, as well as the rate of bleeding. A generally accepted rule is that an output of 200 mL/h or more for 4 hours, or 500 mL of output in any single hour warrants a return trip to the operating room for re-exploration to see if there is any need for surgical hemostasis.

Echocardiography is often technically limited in the immediate postoperative period. In addition, cardiac tamponade in the postsurgical patient is a challenging diagnosis to make by echocardiography. An isolated clot can occur behind the heart and cause tamponade, and this is extremely difficult to visualize with an ECG.

#### **REFERENCE (32)**

St. Andre AC, DelRossi A. Hemodynamic management of patients in the first 24 hours after cardiac surgery. *Crit Care Med* 2005;33:2082-2093.

# SECTION 3: Endocrine/Metabolism

## **SECTION 3: ENDOCRINE/METABOLISM**

**Instructions:** For each question, select the most correct answer.

- 1. Which one of the following is not true concerning calculation and use of the delta gap in patients with an anion gap acidosis?
  - A. The delta gap allows detection of anion gap plus nonanion gap metabolic acidosis
  - B. The use of the delta gap allows identification of a coexisting anion gap metabolic alkalosis
  - C. If an anion gap acidosis is the only metabolic acid base disorder present, the delta gap is expected to be 8-12
  - D. The delta gap requires calculation of the anion gap
- 2. A 45-year-old with severe nephrotic syndrome is admitted with nausea, fever, and hypotension. Her vital signs are BP 88/50 mm Hg, HR 110/min, RR 20/min, temperature 38.3°C (101°F), pH 7.35, Paco<sub>2</sub> 32 mm Hg, sodium 132 mmol/L, potassium 4.0 mmol/L, chloride 103 mmol/L, HCO<sub>3</sub> 17 mmol/L, albumin 1.5 g/dL, blood urea nitrogen 20 mg/dL, and creatinine 1.4 mg/dL.

Which one of the following acid-base disorders is present?

- A. Anion gap metabolic acidosis
- B. Nonanion gap metabolic acidosis
- C. Nonanion gap metabolic acidosis and respiratory alkalosis
- D. Anion and nonanion gap metabolic acidosis

3. A 22-year-old 60-kg female with type 1 diabetes is admitted to the ICU with diabetic ketoacidosis and an altered mental status. Her initial arterial pH was 7.04, anion gap 24, bicarbonate 10 mmol/L, and serum glucose was 420 mg/dL. She received 3 L of normal saline solution and 10 U of regular insulin intravenously. An insulin infusion was started at 6 U/h. After 2 hours, her laboratory results showed pH 7.22, sodium 143 mmol/L, potassium 4 mmol/L, chloride 118 mmol/L, bicarbonate 8 mmol/L, and glucose 65 mg/dL. Her mental status has improved and urine output is 2 mL/kg/h.

Which one of the following is the most appropriate intervention?

- A. Continue insulin infusion at 6 U/h and 0.9% saline solution at 500 mL/h
- B. Change to 0.45% saline solution at 250 mL/h, continue insulin infusion at 6 U/h, and start 10% glucose infusion
- C. Change to 0.45% saline solution at 250 mL/h and decrease insulin infusion to 2 U/h
- D. Continue 0.9% saline solution at 250 mL/h and decrease insulin infusion to 2 U/h
- E. Change to 0.45% saline solution, administer 6 U regular insulin subcutaneously and discontinue insulin infusion in 2 hours
- 4. Diarrhea associated with enteral nutrition is least likely to be caused by which one of the following?
  - A. Dumping syndrome
  - B. Bolus feeding
  - C. Intact whole protein enteral feeding
  - D. Semi-elemental enteral feeding
  - E. Enteral feeding without fiber

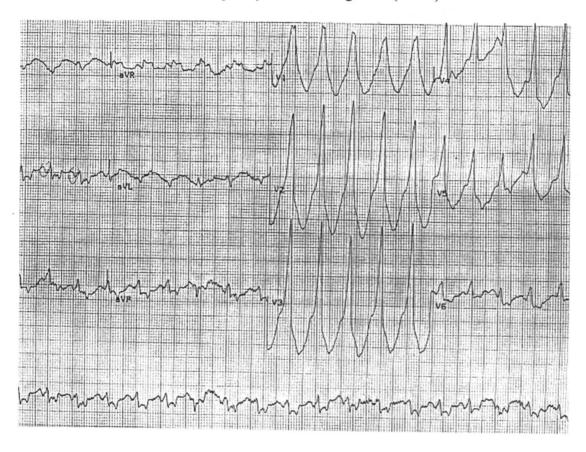
- 5. Which of the following parameters of adrenal function has been most clearly associated with increased mortality in patients with vasopressor-dependent septic shock?
  - A. Random serum total cortisol level  $<34 \mu g/dL$
  - B. Rise in serum total cortisol level >10  $\mu$ g/dL in response to an adrenocorticotropic hormone stimulation test
  - C. Rise in serum total cortisol level <10 µg/dL in response to an adrenocorticotropin hormone stimulation test
  - D. Random serum-free cortisol <5 μg/dL
- 6. Which of the following statements about intensive insulin therapy and glycemic control in critical illness is most correct?
  - A. The survival benefit previously demonstrated with intensive insulin therapy appears to have been a glycemia-independent function of high-dose insulin, rather than an effect of achieving normoglycemia
  - B. In a surgical critical care population, intensive insulin therapy has no demonstrable impact on the development of infection
  - C. Intensive insulin therapy is a stimulus of the pro-inflammatory response
  - D. Protection of endothelial integrity has been demonstrated with intensive insulin therapy and is one of the proposed mechanisms for improved organ function
- 7. Taking a physical-chemical approach to acid-base problems in the ICU, all of the following are independent variables that regulate blood pH except for which one?
  - A. Strong ion difference
  - B. Partial pressure of carbon dioxide (Paco<sub>2</sub>)
  - C. Hydrogen ions (H<sup>+</sup>)
  - D. Weak acids (ie, proteins and phosphates)

- 8. Which of the following statements about hypoglycemia in the ICU is most correct?
  - A. The typical clinical manifestations of hypoglycemia are usually easy to detect in the ICU setting
  - B. When hypoglycemia does occur in the ICU, serious neurologic sequelae are common
  - C. Administering intramuscular glucagon is an effective method of correcting hypoglycemia in the malnourished critically ill patient
  - D. Hypoglycemia can cause focal neurologic deficits
- 9. A 36-year-old woman presents to the emergency department with dyspnea. Three weeks ago she developed the onset of fatigue, dyspnea on exertion, diplopia, and dysphagia. She has difficulty raising her arms above her head, and cannot comb her hair. Over the past 24 hours, she developed resting dyspnea and orthopnea.

On examination she is awake and follows commands. She speaks very softly. She has no fever. Her RR 30/min and shallow. All other vital signs are normal. There is bilateral ptosis and marked proximal muscle weakness in the extremities. Her vital capacity is measured at 600 mL (her ideal body weight is 75 kg). Serum potassium, magnesium, and phosphate levels are normal. Which of the following next steps would be most correct?

- A. Administer steroids
- B. Administer pyridostigmine
- C. Administer immunoglobulin
- D. Obtain arterial blood gases
- E. Endotracheal intubation

10. A 32-year-old woman with type 1 diabetes mellitus presents to the emergency room as unresponsive. Vital signs were BP 114/70 mm Hg, HR 120/min, RR 22/min, and afebrile. An arterial blood gas revealed apH 7.1, Pco<sub>2</sub> 10 mm Hg, PO<sub>2</sub> 180 mm Hg (on supplemental oxygen). Initial laboratory results were sodium 139 mmol/L, potassium 2.5 mmol/L, chloride 105 mmol/L, HCO<sub>3</sub> 5 mmol/L, and glucose 568 mg/dL. Her initial ECG was normal, except for a tachycardia at 120/min. Subsequently, the following ECG (below) was obtained.



What is the most likely event that occurred prior to this ECG?

- A. Bolus of 2 L normal saline solution
- B. Administration of 150 mmol sodium bicarbonate
- C. Administration of a  $\beta$ -blocker
- D. Myocardial ischemia
- E. Administration of 10 mmol potassium phosphate

## **SECTION 3: ENDOCRINE/METABOLISM**

**ANSWERS:** 

1-C; 2-A; 3-B; 4-D; 5-C; 6-D; 7-C; 8-D; 9-E; 10-B

RATIONALE (1)

Answer: C

If an anion gap acidosis is the only metabolic acid base disorder present, then the delta gap should closely approximate the decrease in the serum bicarbonate. Thus, if the expected (normal) anion gap is 10 and the calculated anion gap is 18, then the anticipated serum bicarbonate would be 16 (24-8). There is no "expected" delta gap, because it should vary based on the degree of anion gap acidosis, and equal the drop in bicarbonate in a pure anion gap acidosis. The delta gap not only allows the detection of a coexisting nonanion gap metabolic acidosis, such as that present in chronic respiratory alkalosis (if the anticipated serum bicarbonate is less than the anion gap, acidosis would have been predicted), but it also allows detection of a coexisting metabolic alkalosis. This is ascertained when the serum bicarbonate is greater than what was anticipated based on the delta gap. This frequently occurs in diabetic ketoacidosis (DKA) patients in whom DKA was preceded by nausea and vomiting and a contraction alkalosis. The use of the delta gap in this circumstance allows the diagnosis of DKA.

#### REFERENCES (1)

Brink SJ. Diabetic ketoacidosis. Acta Paediatr Suppl 1999;427:14-24.

Kitabchi AE, Umpierrez GE, Murphy MB, et al. Management of hyperglycemic crises in patients with diabetes. *Diabetes Care* 2001;24:131-153.

RATIONALE (2)

Answer: A

In this patient, the pH of 7.35 suggests an acidosis, and the low bicarbonate along with decreased  $Paco_2$  suggest a metabolic process. The respiratory compensation is appropriate, using the formula  $Paco_2 = 1.5 \text{ x } [Hco_3] + 8 \pm 2$ . The anion gap is usually calculated as sodium – (Cl + [Hco\_3]), which in this patient yields an anion gap of 12 mmol/L. The normal anion gap is assumed to be 10-12 mmol/L for this question.

An anion gap of 12 might be considered normal for this patient, except that the albumin concentration must be taken into account. The normal range for the anion gap is decreased in hypoalbuminemic patients. For every decrease of 1 g/dL in albumin, a decrease of 2.5 to 3 mmol/L in the anion gap occurs. In this patient, the normal anion gap is closer to 5 or 6 mmol/L. Therefore, an anion gap acidosis exists.

Correction of the anion gap for phosphate levels has been suggested by some, but it will not usually impact interpretation of an acid-base disorder. The clinical scenario in this case suggests infection and/or hypotension as the cause of the acidosis. Answers (B) and (C) are incorrect, because an anion gap metabolic acidosis exists, and the respiratory alkalosis is an appropriate compensation rather than a primary disorder. Answer (D) is incorrect since the decrease in serum bicarbonate is similar to the increase in anion gap (referred to as the delta gap).

#### **REFERENCES (2)**

Fencl V, Jabor A, Kazda A, Figge J. Diagnosis of metabolic acid-base disturbances in critically ill patients. *Am J Respir Crit Care Med* 2000;162:2246-2251.

Figge J, Jabor A, Fencl V. Anion gap and hypoalbuminemia. *Crit Care Med* 1998;26:1807-1810. Paulson WD. How to interpret the anion gap. *J Crit Illness* 1997;12:96-99.

Salem MM, Mujais SK. Gaps in the anion gap. Arch Intern Med 1992;152:1625-1629.

Wrenn K. The delta (D) gap: an approach to mixed acid-base disorders. *Ann Emerg Med* 1990;19:1310-1313.

RATIONALE (3)

Answer: B

In this patient, because there is still an anion gap present and the pH is still <7.25, the insulin infusion should be continued at a dose of 6 U/h (0.1 U/kg/h), with 10% glucose initiated to maintain the serum glucose level. The insulin infusion should be continued at the recommended dose until the anion gap acidosis has resolved. It can be decreased to 0.05 U/kg if the pH was ≥7.25. It is not appropriate to decrease or stop the insulin infusion as long as an anion gap is present.

This patient also demonstrates the development of a hyperchloremic state due to an infusion of 0.9% saline solution. After the initial 3 L of 0.9% saline solution with clinical assessment of restoration of adequate intravascular volume, it is appropriate to change to a solution containing less chloride. Although a hyperchloremic metabolic acidosis may have little impact clinically, the persistence of a low pH may mislead the clinician on the status of resolving ketoacidosis. The amount of fluid administration should be guided by the patient's clinical status, utilizing vital signs and urine output. Subcutaneous insulin should not be initiated until the anion gap acidosis is resolved. At that time, an initial dose of subcutaneous insulin should be given, and the insulin infusion continued for 2 to 3 hours to prevent recurrence of acidosis.

#### **REFERENCES (3)**

Delaney MF, Zisman A, Kettyle WM. Diabetic ketoacidosis and hyperglycemic hyperosmolar nonketotic syndrome. *Endocrin Metab Clin North Am* 2000;29:683-705.

Kitabchi AE, Umpierrez GE, Murphy MB, et al. Management of hyperglycemic crisis in patients with diabetes. *Diabetes Care* 2001;24:131-153.

Stoller W, Mazzone T. Acute diabetic emergencies and hypoglycemia. In Parrillo JE, Dellinger RP, eds. *Critical Care Medicine*, 2nd ed. St. Louis, MO: Mosby; 2001:1204-1224.

RATIONALE (4)

Answer: D

Semi-elemental enteral nutrition contains hydrolyzed protein as the nitrogen source, as compared with amino acids in elemental diets, and whole protein in intact protein feeding. These semi-elemental or hydrolyzed protein enteral feeding products are absorbed in the proximal small bowel, and minimal residue is transferred to the colon, thereby reducing diarrhea. This type of feeding is also advantageous for early feeding in trauma and postoperative patients. It is thought that secretory diarrhea is reduced.

Diarrhea complicates the course of one third of critically ill patients. Investigation of the cause of diarrhea includes examining medications, infections, electrolytes, and the type of enteral nutrition. The use of enteral nutrition at high volumes or with high osmolarity increases the chance of diarrhea. Elemental formulas are hypertonic and can cause diarrhea, nausea, vomiting, and abdominal distension.

Dumping syndrome is caused by the rapid emptying of the hypertonic gastric contents into the small bowel. The distension of the small bowel releases neurohumoral factors that cause vasomotor and gastrointestinal symptoms (bloating and diarrhea). A carbohydrate load (enteral feeding with high carbohydrates) stimulates this problem. Diarrhea secondary to dumping syndrome is controlled by reducing the carbohydrate content and increasing the fat and protein content of the enteral diet.

Bolus feeds may stimulate the dumping syndrome from gastrointestinal intolerance to carbohydrates, protein, or fat. In patients with albumin <2.5 g/dL, gut absorption is impaired, and whole protein formula will not be tolerated. Elemental or semi-elemental formulas should be used. Fiber reduces stool water content and improves stool consistency. Control of diarrhea is achieved by using a semi-elemental formula, then adding increasing doses of bismuth subsalicylate and loperamide hydrochloride).

#### **REFERENCES (4)**

- Bowling TE. Enteral feeding related diarrhea: proposed causes and possible solutions. *Proc Nutr Soc* 1995;54:579-590.
- Dobb CJ, Towler SC. Diarrhea during enteral feeding in the critically ill: a comparison of feeds with and without fiber. *Int Care Med* 1990;16:252-255.
- Hwang TL, Lue MC, Nee YL, et al. The incidence of diarrhea in patients with hypoalbuminemia due to acute or chronic malnutrition during enteral feeding. *Am J Gastroenterol* 1994;89:376-381.
- Meredith JW, Ditesheim JA, Zaloga GP. Visceral protein levels in trauma patients are greater with peptide diet than intact protein diet. *J Trauma* 1990;30:825-833.

RATIONALE (5)

Answer: C

Work from Annane and colleagues has identified that relative adrenal insufficiency is common in patients with vasopressor-dependent septic shock. This adrenal dysfunction can be prognostic in terms of mortality risk. Although the absolute value for a random serum total cortisol level may have some prognostic implications in the setting of vasopressor dependency, the most important predictor appears to be the ability to increase serum total cortisol in response to an adrenocorticotropin hormone stimulation test. Failure to increase cortisol by  $10~\mu g/dL$  or more in response to an adrenocorticotropin hormone stimulation test identifies a high risk of death in patients with vasopressor-dependent septic shock. Although cortisol is mostly protein bound, and protein deficiency can impact cortisol measurement, the role of measuring free cortisol in septic shock patients has not yet been well defined.

#### **REFERNCES (5)**

Annane D, Sebille V, Troche G, Raphael JC, Gajdos P, Bellissant E. A 3-level prognostic classification in septic shock based on cortisol levels and cortisol response to corticotropin. *JAMA* 2000;283:1038-1045

Salvatori R. Adrenal insufficiency. JAMA 2005;294:2481-2488.

RATIONALE (6)

Answer: D

The work of van den Berghe and colleagues has identified that intensive insulin therapy in the ICU setting may have a significant outcome benefit. In a population of mechanically ventilated surgical critical care patients with heterogeneous diagnoses, intensive insulin therapy was associated with a mortality benefit. The survival benefit that has been associated with intensive insulin therapy appears to be a function of maintaining normoglycemia rather than an effect of the insulin dose itself.

In theory, tight glycemic control can improve phagocytic activity and bacterial killing, and intensive insulin therapy has been associated with a demonstrable impact on the development of sepsis in the critically ill. Intensive insulin therapy has also been associated with an antiinflammatory effect. One other proposed mechanism of the action of tight glycemic control is the protection of endothelial integrity, which could, in theory, have an impact on modulating organ function and dysfunction.

It is important to remember that intensive insulin therapy has been shown to improve survival in a select population (surgical critical care). In fact, intensive insulin therapy has not been associated with a robust treatment effect in one study in a medical ICU population. Whether or not the benefits of intensive insulin therapy can be generalizable to heterogenous populations of critically ill patients in a multidisciplinary ICU setting is currently unknown.

#### **REFERENCES (6)**

Van den Berghe G, Wilmer A, Hermans G, et al. Intensive insulin therapy in the medical ICU. N Engl J Med 2006;354:449-461.

Van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med 2001;345:1359-1367.

RATIONALE (7)

Answer: C

An alternative approach to deciphering acid-base derangements in the ICU has been recently advocated in the critical care literature. In contrast to conventional acid-base teaching, which is grounded in the Henderson-Hasselbach equation, a physical-chemical approach to acid-base problems was first introduced by Peter Stewart more than two decades ago. In this conceptual framework, the physical-chemical approach to acid-base analysis can lead to an improved understanding of the mechanisms behind acid-base disorders and why they occur. Specifically, it facilitates recognition of the way that fluid therapy administered in the ICU may modulate acid-base derangements.

The strong ion difference (SID) is the balance of net charges between all strong ions (i.e., completely dissociated) that are present. The strong anions include chloride and lactate. The strong cations include sodium, potassium, calcium, and magnesium. Although some unmeasured ions may also be present, the SID is the sum of the strong cations minus the sum of the strong anions. Usually this is around 40 mEq/L in a normal host, but can be markedly abnormal in a critically ill patient. Narrowing of the strong ion difference indicates worsening acidosis. This helps us to understand why massive fluid resuscitation with normal saline (ie, sodium chloride) worsens or induces acidosis, as the high chloride content narrows the SID.

The other two variables (besides the SID) that regulate blood pH include the total concentration of "weak acids" (mostly proteins and phosphates) and Pco<sub>2</sub>. The Stewart physical-chemical approach is based on the understanding that only these three variables are important, as hydrogen ion concentration and Hco<sub>3</sub> cannot change unless one of those three variables changes. This approach also takes into account the important role of plasma proteins, most notably albumin, in acid-base derangements. Clearly the traditional approach of focusing on the anion gap can be problematic in the setting of hypoalbuminemia, because the normal anion gap would be much lower than expected.

In summary, the Stewart physical-chemical approach to acid-base balance, focusing on a strong ion difference represents an alternative to approaching acid-base derangements in the ICU and may provide insights that are meaningful at the bedside.

#### **REFERENCES (7)**

Gunnerson KJ, Kellum JA. Acid-base and electrolyte analysis in critically ill patients: are we ready for the new millennium? Curr Opin Crit Care 2003;9:468-473

Kellum JA. Acid-base physiology in the post-Copernican era. Curr Opin Crit Care 1999, 5:429-435. Gunnerson KJ, Kellum JA. Acid-base and electrolyte analysis in critically ill patients: are we ready for the new millennium? Curr Opin Crit Care 2003;9:468-473

RATIONALE (8)

Answer: D

Hypoglycemia (even transient in duration) can cause profound neurological changes, including focal neurological deficits. If hypoglycemia is not rapidly identified and corrected, severe neurologic sequela can occur. Fortunately, serious neurological sequela are uncommon in the ICU setting, perhaps due to the fact that the close monitoring of a critical care unit allows for the rapid identification and correction of the blood sugar. Nonetheless, hypoglycemia is a common occurrence in ICU populations. Because ICU patients are often mechanically ventilated or neurologically compromised, the typical manifestations of hypoglycemia (especially the subjective symptomatology) could potentially go unrecognized. Therefore, frequent blood glucose monitoring is necessary in patients at risk for hypoglycemia (i.e., poor nutritional status, hepatic failure, and diabetics treated with insulin).

The conventional way to treat hypoglycemia in the ICU is with an intravenous infusion of dextrose. Although glucagon has been utilized to correct hypoglycemia in the prehospital setting, especially when intravenous access has not yet been established, this may not be an effective method for correcting hypoglycemia in the critically ill. Glucagon achieves an increase in blood sugar by releasing glucose from glycogen stores in the liver. Patients with critical illness, especially prolonged critical illness, may have depleted glycogen stores and may not respond appropriately to glucagon.

#### **REFERENCES (8)**

Vriesendorp TM, Devries JH, van Santen S, et al. Evaluation of short-term consequences of hypoglycemia in an intensive care unit. *Crit Care Med* 2006;34:2714-2718.

Vriesendorp TM, van Santen S, DeVries JH, et al. Predisposing factors for hypoglycemia in the intensive care unit. *Crit Care Med* 2006;34:96-101.

RATIONALE (9)

Answer: E

This patient presents with a myasthenic crisis. Her vital capacity (<10 mL/kg) is indicative of a neuromuscular ventilatory failure. An arterial blood gas analysis is not necessary in order to confirm that she is in respiratory failure and requires mechanical ventilation in order to prevent her from further respiratory decompensation. All of the other options (A-D) represent potential therapies for a patient with a myasthenic crisis, but the most correct next action is to initiate ventilatory support.

#### **REFERENCES (9)**

- De Jonghe B, Lacherade JC, Durand MC, Sharshar T. Critical illness neuromuscular syndromes. *Crit Care Clin*. 2007; 23:805-818.
- De Jonghe B, Lacherade JC, Durand MC, Sharshar T. Critical illness neuromuscular syndromes. *Crit Care Clin*. 2006; 22:508-818.
- Dhand UK. Clinical approach to the weak patient in the intensive care unit. Respir Care. 2006; 51:1024-40.
- Gorson KC. Approach to neuromuscular disorders in the intensive care unit. *Neurocrit Care*. 2005; 3:195-212.
- Maramattom BV, Wijdicks EF. Acute neuromuscular weakness in the intensive care unit. *Crit Care Med.* 2006; 34:2835-3841.
- Mehta S. Neuromuscular disease causing acute respiratory failure. Respir Care. 2006; 51:1016-1021.

RATIONALE (10)

Answer: B

The ECG shows a wide-complex tachycardia. Although myocardial ischemia is always a consideration in a diabetic patient, the most likely reason for the development of these ECG changes is a decrease in her serum potassium level due to the administration of sodium bicarbonate. Sodium bicarbonate would result in transcellular movement of potassium into the cell. In diabetic ketoacidosis, there is no documented benefit of the use of sodium bicarbonate, and it is not recommended in patients with pH>7.0. If sodium bicarbonate is used, it should be administered as an infusion, not a bolus.

Additional complications that can be associated with sodium bicarbonate administration include hypocalcemia, hypernatremia, volume overload, respiratory acidosis, intracellular acidosis, and hyperosmolarity. This patient requires potassium replacement before any administration of insulin, as insulin can result in a similar decrease in serum potassium. Potassium should be repleted before administering insulin if the potassium level is less than 3.3 mmol/L.

Saline administration is unlikely to result in electrolyte changes that would precipitate the ECG changes. Potassium phosphate would treat the deficiency. A  $\beta$ -blocker is also unlikely to cause these ECG changes.

#### **REFERENCES (10)**

- American Diabetes Association. Hyperglycemic crises in patients with diabetes mellitus-position statement. *Diabetes Care* 2003;26:S109-S117.
- Forsythe SM, Schmidt GA. Sodium bicarbonate for the treatment of lactic acidosis. *Chest* 2000;117:260-267.
- Viallon A, Zeni F, Lafond P, et al. Does bicarbonate therapy improve the management of severe diabetic ketoacidosis? *Crit Care Med* 1999;27:2690-93.

# SECTION 4: Gastrointestinal

## **SECTION 4: GASTROINTESTINAL**

Instructions: For each question, select the most correct answer.

- 1. Which one of the following statements about stress ulcer prophylaxis in the ICU is most correct?
  - A. The incidence of clinically significant bleeding with the use of sucralfate is lower than the incidence of clinically significant bleeding with the use of H<sub>2</sub>-antagonists
  - B. The use of H<sub>2</sub>-antagonists has little impact on the incidence of clinically significant bleeding
  - C. The risk of nosocomial pneumonia is significantly higher, with H<sub>2</sub>-antagonists as compared with the use of sucralfate
  - D. Administration of once-daily proton-pump inhibitors achieves the same elevation of intragastric pH as a continuous infusion of H<sub>2</sub>-antagonists
- 2. What are the risk factors that have the strongest correlation with stress-related bleeding in critically ill patients?
  - A. Mechanical ventilation and glucocorticoids
  - B. Coagulopathy and sepsis
  - C. Mechanical ventilation and coagulopathy
  - D. Sepsis and renal failure
  - E. Mechanical ventilation and hypotension

3. A 45-year-old male with liver cirrhosis secondary to alcohol abuse presents to the emergency room with hematemesis and lightheadedness. Patient is noncompliant with medications and still drinks alcohol. He appears cachectic and pale. On examination, HR is 110/min, BP 100/60 mm Hg, and RR is 20/min. The examination of the heart and lungs is unremarkable; the abdomen is soft, but nontender. There is significant ascites, and large collateral veins are observed on the abdominal wall. Rectal examination is normal. Stool is black and strongly guaiac positive.

Two large bore peripheral IVs are obtained, and aggressive hydration with 0.9% normal saline solution is initiated. The patient is treated with an IV famotidine and an IV octreotide drip. Laboratory data are pending. Patient's hematemesis continues; after admission to the ICU, he becomes lethargic. A decision to intubate the patient and secure the airway is made.

Which one of the following statements is most correct regarding the acute management of this patient?

- A. If emergent endoscopy is not immediately available, a Sengstaken-Blakemore tube is indicated
- B. Immediate angiogram with possible embolization
- C. Sclerotherapy and an esophageal balloon tamponade have comparable efficacy in controlling esophageal bleeding
- D. Transfusion of fresh frozen plasma is indicated prior to procedural interventions
- E. Patient should be stabilized with crystalloids and blood transfusions, pending an anatomic diagnosis of bleeding

4. A 62-year-old male with history of chronic obstructive pulmonary disorder was admitted to the ICU with respiratory failure. The patient has been on mechanical ventilation for 7 days. He is currently receiving antibiotics for pneumonia. He has received intermittent doses of benzodiazepines and narcotics for sedation and analgesia. Over the last 2 days, he has developed a progressive abdominal distention with poor tolerance of enteral feedings secondary to high gastric residuals.

On examination, you notice a distended abdomen with no evidence of surgical scars. There is a significant tympany on percussion and diffuse tenderness on palpation, with no rebound tenderness, guarding or rigidity. There is no evidence of ascites. On auscultation, there is decreased bowel sounds on all quadrants.

An abdominal radiograph (flat plate) is obtained and shows dilatation of the cecum (12 cm) with no other significant findings.

Which one of the following statements is most correct regarding this case?

- A. Surgical intervention is indicated
- B. Central venous alimentation should be initiated and maintained for a period of 2 weeks
- C. Neostigmine is indicated
- D. Atropine is indicated
- E. Colonoscopy is contraindicated

5. An 85-year-old female is admitted to the ICU with septic shock and pneumonia. She requires mechanical ventilation, vasopressor support, and broad-spectrum antibiotics. After 4 days of aggressive support she is afebrile but remains intubated and requiring norepinephrine for BP support. On day 6, the patient develops a low-grade fever and scleral icterus.

Abdominal examination is significant for tenderness on palpation of the right upper quadrant; there is no rebound tenderness, guarding, or rigidity. Bowel sounds are present. Laboratory data show normal electrolytes, blood urea nitrogen, and creatinine. Her white blood cell count has increased from 9,500/mm³ to 12,000/mm³ in 24 hours. aspartate aminotransferase, alanine amino transferase and lactate dehydrogenase are within normal range. Bilirubin is 5 mg/dL, with a direct bilirubin of 3.4 mg/dL.

Bedside abdominal ultrasound shows a dilated gallbladder with thickened walls and biliary sludge with no evidence of stones. No other abnormalities are noted.

Which one of the following is the most correct action for this case?

- A. Continue present management
- B. Add metronidazole to antibiotic regimen and observe patients evolution
- C. Emergent laparotomy
- D. Percutaneous cholecystostomy
- E. Perform a hepatobiliary scintigraphy to confirm diagnosis
- 6. A 75-year-old patient with chronic obstructive pulmonary disorder and atrial fibrillation is admitted to the ICU for resuscitation. The patient complains of severe abdominal pain and has a soft distended tender abdomen. Temperature is 9°C (102.2°F); systolic BP is 90 mm Hg. The white blood cell count is 30,000/µL and the creatinine is 2 mg/dL.

Which one of the following test is most likely to lead to the correct diagnosis?

- A. Chest radiograph
- B. Arteriogram
- C. CT scan of the abdomen
- D. Lower GI endoscopy
- E. Ultrasound of the abdomen

7. A 55-year-old female presents to the emergency room with a 24-hour history of abdominal pain. Diagnostic work up is consistent with acute pancreatitis.

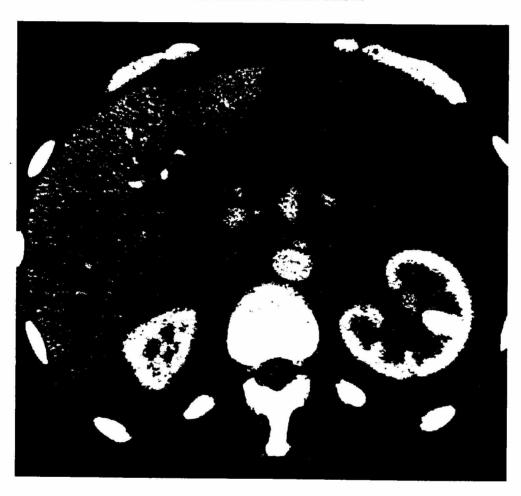
Which of the following findings is most likely to predict complications and the need for intense monitoring and ICU admission?

- A. Abdominal guarding on physical examination
- B. Serum lipase >5,000 U/dL
- C. Acute Physiology and Chronic Health Evaluation (APACHE) II score of 16
- D. A CT scan of the abdomen showing an edematous pancreas
- E. Aspartate aminotransferase >120 U/L

8. You are asked to evaluate a 45-year-old male who presents to the emergency department with a 2-day history of severe epigastric pain, nausea, and vomiting. The patient has a past medical history significant for alcohol abuse and hypertension. On physical examination, vitals signs are as follows: temperature 38.6°C (101.2°F), BP 100/70 mm Hg, HR 110/min, RR of 26/min. Abdominal examination reveals diffused tenderness on palpation, with guarding and absent bowel signs.

Laboratory data are hemoglobin 15.4 g/dL, white blood cells 19,000/ $\mu$ L, amylase 600 U/dL, lipase 5,000 U/dL, lactate dehydrogenase 700 U/dL, creatinine 2.2 mg/dL, and calcium 8.5 mg/dL.

A CT scan of the abdomen with contrast is shown below.



Which of the following statements is most correct regarding the treatment of this patient?

- A. Emergent laparotomy should be performed
- B. Antibiotic treatment with imipenem should be initiated
- C. Total enteral nutrition via nasojejunal feeding tube is recommended for nutritional support
- D. Systemic corticosteroid therapy should be initiated

9. A 30-year-old woman is brought to the hospital by family members after being found unresponsive. The patient has no significant past medical history; however, the family reports that she has been depressed. On physical examination, vital signs are temperature 37.3°C (99.1°F), HR 110/min, RR 25/min, and BP of 110/60 mm Hg.

The patient is lethargic, although she does open her eyes to stimuli. Abdominal examination reveals mild diffuse tenderness, with no guarding and decreased bowel sounds. Laboratory data are significant for the following: alanine amino transferase 3,800 U/L, aspartate aminotransferase 4,300 U/L, total bilirubin 7.5 mg/dL, and INR of 2.5.

Which of the following is the most likely to result in mortality in this patient?

- A. Uncontrolled bleeding
- B. Septic shock
- C. Brain edema
- D. Respiratory failure
- E. Acute myocardial infarction
- 10. Which one of the following statements is most correct concerning severe pancreatitis?
  - A. The diagnosis of pancreatic necrosis is best made by ultrasound examination
  - B. Fine needle aspiration of necrotic areas of the pancreas, guided by either a CT scan or ultrasound, is a reliable method of identifying infected necrosis
  - C. Enteral feeding should be avoided in patients with severe complicated pancreatis
  - D. Surgical intervention for patients with pancreatic necrosis is best performed within the first few days after onset
- 11. Which one of the following is most correct concerning acute acalculous cholecystitis?
  - A. The majority of patients who develop acute acalculous cholecystitis are hospital inpatients
  - B. The age of onset of patients with acute acalculous cholecystitis is younger than patients with calculous cholecystitis
  - C. Acalculous cholecystitis is more common in men
  - D. Ultrasound better defines pericholecystic inflammation than does CT scan

12. You are asked to evaluate a 50-year-old obese female for admission to your ICU. She is known to have had a solitary asymptomatic gallstone for 10 years. This morning she developed severe right upper quadrant pain. She is febrile to 38.7°C (101:6°F) and has marked scleral icterus. She reports a shaking chill earlier in the day. BP is 90/60 mm Hg, HR is 120/min, and RR is 24/min. The patient has not voided since last evening, and a catheterized urine specimen is positive for bilirubin, negative for white blood cells (WBCs), and yields a total volume of 100 mL. Her WBC count is 16,000/mm³, with a marked shift to the left. Direct bilirubin is 6.8 mg/dL.

On physical examination, she is tender over the right upper quadrant without masses palpated. No rebound tenderness is appreciated. An emergency ultrasound performed 1 hour ago fails to reveal either gallstones or dilation of the intrahepatic ducts.

Which one of the following represents the most correct initial management decision for this patient?

- A. Admit to the ICU for observation, IV hydration, and antibiotic therapy
- B. Immediate endoscopic retrograde cholangiopancreatography
- C. Immediate percutaneous transhepatic drainage
- D. Immediate surgical exploration

### **SECTION 4: GASTROINTESTINAL**

**ANSWERS:** 

1-D; 2-C; 3-E; 4-C; 5-D; 6-B; 7-C; 8-C; 9-C; 10-B; 11-C, 12-A

RATIONALE (1)

Answer: D

Stress-related mucosal disease in the ICU results in clinically significant gastrointestinal bleeding and increased mortality. Although many aspects of critical illness (including shock, sepsis, organ dysfunction, etc) may increase the risk of gastrointestinal bleeding, the only risk factors found to achieve statistical significance were coagulopathy and mechanical ventilation. In patients already receiving mechanical ventilation, renal failure, the absence of enteral nutrition, and the withholding H<sub>2</sub>-antagonists were shown to be independent predictors of clinically significant bleeding.

 $\rm H_2$ -antagonists and sucralfate have both been shown to reduce the risk of clinically significant bleeding. In the 1998 study by Cook and colleagues, ranitidine was more effective at decreasing the risk of bleeding than sucralfate. Once-daily proton-pump inhibitors can achieve the same rise in intragastric pH as a continuous infusion of  $\rm H_2$ -antagonists.

Increasing intragastric pH may increase the risk of nosocomial pneumonia. There is an increased incidence of pneumonia when the use of  $H_2$ -blockers is compared with no prophylaxis. However, in the largest trial to date, there was no significant difference in the rate of nosocomial pneumonia when  $H_2$ -antagonists were compared with sucralfate.

Although the studies of clinically significant bleeding with proton-pump inhibitor prophylaxis would suggest a decreased risk of bleeding, the studies are limited by relatively small sample size as compared with previous investigations in SRMD.

#### **REFERENCES (1)**

- Cash BD. Evidence-based medicine as it applies to acid suppression in the hospitalized patient. *Crit Care Med.* 2002; 30:S373–S378.
- Cook D, Guyatt G, Marshall J, et al. A comparison of sucralfate and ranitidine for the prevention of upper gastrointestinal bleeding in patients requiring mechanical ventilation: Canadian Critical Care Trials Group. N Engl J Med. 1998; 338:791-797.
- Cook DJ, Reeve BK, Guyatt GH, et al. Stress ulcer prophylaxis in critically ill patients. Resolving discordant meta-analyses. *JAMA*. 1996; 275:308-314.
- Cook DJ, Fuller HD, Guyatt GH, et al. Risk factors for gastrointestinal bleeding in critically ill patients: Canadian Critical Care Trials Group. N Engl J Med. 1994; 330:377-381.
- Steinberg K. Stress-related mucosal disease in the critically ill patient: Risk factors and strategies to prevent stress-related bleeding in the intensive care unit. Crit Care Med. 2002; 30:S362-S364.

RATIONALE (2)

Answer: C

The risk of developing stress-related gastrointestinal bleeding varies in critically ill patients. It is important to recognize patients with increased risk factors in order to develop cost-effective prophylactic regimens. In a landmark study on risk factors, Cook et al found that the strongest independent risk factors for the development of stress-related bleeding in ICU patients were mechanical ventilation for >48 hours (RR 16/min, p <0.01) and coagulopathy (RR 4.3/min, p <0.01). Coagulopathy was defined as a platelet count <50,000/mL, prothrombin time greater than 1.5 times control value, or a partial thromboplastin time greater than 2 times the control value.

Hypotension was found to have an increased odds ratio (3.7), but did not achieve statistical significance. Renal failure, sepsis, liver failure, enteral feeding, and glucocorticoids did not achieve statistical significance. In this study the incidence of clinically significant stress-related bleeding was 3.7% in patients with risk factors compared to 0.1% in patients without risk factors.

#### **REFERENCES (2)**

- Cook DJ, Fuller HD, Guyatt GH, et al Risk factors for gastrointestinal bleeding in critically ill patients: Canadian Critical Care Trials Group. N Engl J Med 1994;330:377-381.
- Stainberg K: Stress-related mucosal disease in the critically ill patients: risk factors and strategies to prevent stress-related bleeding in the intensive care unit. Crit Care Med 2002;30:S362-S365.

RATIONALE (3)

Answer: E

The patient should be stabilized with aggressive crystalloid resuscitation and the transfusion of blood products as needed. Once this is accomplished, or if the patient has persistent bleeding, emergent endoscopy should be performed in order to establish an anatomical diagnosis and attempt a hemostasis of bleeding lesion.

An esophageal balloon tamponade with a Sengstaken-Blakemore tube is indicated in cases of confirmed esophageal variceal hemorrhage in which endoscopic therapy is unavailable, technically not feasible or unsuccessful. Placement of a Sengstaken-Blakemore tube should not be performed without a firm anatomical diagnosis. Although the patient in this case is at high risk for esophageal variceal hemorrhage, severe gastrointestinal bleeding in patients with signs of chronic liver disease can result from other causes in up to 35% of cases.

Endoscopic therapy with sclerotherapy or banding has been demonstrated to be more effective than an esophageal balloon tamponade in treating acute esophageal variceal hemorrhage.

Without documentation of coagulopathy, there is no reason to transfuse fresh frozen plasma in this patient.

#### **REFERENCES (3)**

Hegab AM, Luketic VA. Bleeding esophageal varices. How to treat this dreaded complication of portal hypertension. *Postgrad Med* 2001;109:81-86.

Pasquale MD, Cerra FB. Sengtaken-Blakemore tube placement. Use of the balloon tamponade to control bleeding varices. *Crit Care Clin* 1992;8:743-753.

Stiegmann GV, Goff JS, Michaletz-Onody PA, et al. Endoscopic sclerotherapy as compared with endoscopic ligation for bleeding esophageal varices. *N Engl J Med* 1992;326:1527-1532.

RATIONALE (4)

Answer: C

The patient's presentation is consistent with acute colonic pseudo-obstruction, also known as Ogilvie syndrome. Ogilvie syndrome is characterized by gross dilation of the colon, usually the cecum, in the absence of an anatomic obstructing lesion. The small intestine might also be dilated in some cases. This syndrome can be seen in critically ill patients admitted to the ICU or long-term acute care centers. Risk factors include electrolyte abnormalities, use of antimotility agents and narcotics. The exact mechanism that produces colonic atony remains unclear. However, autonomic nervous dysfunction is thought to play an important role.

Clinical presentation usually includes abdominal distension, tympany, diffuse tenderness, and reduced bowel sounds. Plain radiographs will reveal a dilated colon, most often cecum. Complications as ischemia and perforation are uncommon with cecal diameters of less than 12 cm. The rate of development and the duration of dilation probably relate better to the rate of complications. Management involves supportive measures, with treatment of the underlying disease and the correction

of any electrolyte abnormalities. Gastrointestinal decompression with nasogastric and rectal tubes can be used.

Studies have demonstrated the effectiveness of neostigmine (2 mg IV) in producing rapid decompression and preventing recurrence. Neostigmine is well tolerated in general but may produce bradycardia, which is responsive to atropine.

Cases refractory to conservative and medical treatment may require colonoscopy for decompression. Colonoscopy in the ICU patient with an unprepped bowel has a high complication rate and should be avoided, if possible. Surgery is indicated in patients where other therapeutic modalities fail or in those who develop complications such as ischemia and perforation. Initiation of central venous alimentation is not indicated in patients with prompt response to therapy.

#### **REFERENCES (4)**

Ponec RJ, Saunders MD, Kimmey MB. Neostigmine for the treatment of acute colonic pseudo-obstruction. *N Engl J Med* 1999;341:137-140.

Seth SG, LaMont JT. Gastrointestinal problems in the chronically critically ill patient. *Clin Chest Med* 2001;22:135-147.

Stephenson BM, Morgan AR, Salaman JR, et al. Ogilvie's syndrome: a new approach to an old problem. *Dis Colon Rectum* 1995;38:422-427.

Turegano-Fuentes F, Munoz-Jimenez F, Del Valle-Hernandez E, et al. Early resolution of Ogilvie's syndrome with intravenous neostigmine: a simple, effective treatment. *Dis Colon Rectum* 1997;40:1353-1357.

RATIONALE (5)

Answer: D

This patient has acute acalculous cholecystitis (AAC); a percutaneous cholecystostomy is indicated. ACC is a complication recognized in critically-ill patients, with a reported incidence of approximately 1 to 3%. ACC is defined as an acute inflammation of the gallbladder in the absence of stones. Risk factors associated with ACC include mechanical ventilation (>72 hours), shock, sepsis multiple transfusions, and total parenteral nutrition.

The underlying pathophysiology is most likely multifactorial, with ischemia and chemical injury playing a critical role. Diagnosis usually is delayed, due to the inability of patients to express symptoms and the lack of specific biochemical markers. It commonly presents with right upper quadrant/ abdominal pain (which may be masked by the use of sedatives and narcotics), an unexplained fever, leucocytosis, and an increased bilirubin with no other alterations in liver enzymes. Imaging techniques provide further support for diagnosis.

Currently, abdominal ultrasound is the screening test of choice. Findings on ultrasound that are strongly suggestive of ACC include gallbladder distention, gallbladder wall thickening, and the presence of biliary sludge. Pericholecystic fluid collections can be seen, and may be associated with gallbladder perforation. A CT scan is more sensitive than ultrasound and is superior in the evaluation for other

potential diagnoses. However, the availability for bedside examination in this subset of critically-ill patients makes an abdominal ultrasound a more appealing first line test.

Hepatobiliary scintigraphy has a high rate of false positives in critically-ill patients and is not useful for confirming the diagnosis.

Treatment includes supportive care, antibiotics, and drainage. Percutaneous drainage is effective and can be safely done in critically ill patients. Although cholecystectomy has been the traditional approach, the risk of morbidity and mortality with emergent laparotomy in a patient requiring vasopressors and mechanical ventilation make percutaneous drainage a better option. Without gallbladder drainage, there is a significant risk of perforation and peritonitis.

#### **REFERENCES (5)**

Batey A, Khan MA. Acalculous cholecystitis. Clin Gastroenterol Hepatol 2007;5:e8.

Laurila J, Laurila PA, Saarnio J, Koivukangas V, Syrjala H, Ala-Kokko TI. Organ system dysfunction following open cholecystectomy for acute acalculous cholecystitis in critically ill patients. *Acta Anaesthesiologica Scandinavica* 2006;50:173-179.

Mirvis SE, Vainright JR, Nelson AW, et al. The diagnosis of acute acalculous cholecystitis: a comparison of sonography, scintigraphy, and CT. Am J Roentgenol 1986;147:1171-1175.

Molenat F, Boussuges A, Valantin V, et al. Gallbladder abnormalities in medical ICU patients: an ultrasonographic study. *Intensive Care Med* 1996;22:356-358.

Mutlu G, Mutlu EA, Factor P. GI complications in patients receiving mechanical ventilation. *Chest* 2001;119:1222-1241.

Owen CC, Jain R. Acute Acalculous Cholecystitis. *Curr Treat Options Gastroenterol* 2005;8:99-104. Seth SG, LaMont JT: Gastrointestinal problems in the chronically critically ill patient. *Clin Chest Med* 2001;22:135-147.

RATIONALE (6)

Answer: B

This patient has mesenteric ischemia; the radiographic study of choice is an anterior posterior and lateral arteriogram. The classic presentation of acute mesenteric ischemia is an elderly patient with a cardiac arrhythmia and pain out of proportion to the physical findings. Unfortunately, multiple medical problems exist which interfere with the prompt diagnosis of acute mesenteric ischemia.

In an ICU setting, persistent lactic acidosis that fails to respond to aggressive fluid resuscitation points toward this diagnosis. Risk factors include valvular heart disease, congestive heart failure, cardiac arrhythmias (particularly atrial fibrillation), and a recent myocardial infarction. Embolism is responsible for one third of all mesenteric vascular occlusions. In 50% of cases, the emboli lodge in the superior mesenteric artery (SMA), close to the take off of the middle colic artery. The origin of the SMA is occluded in about 15% of cases.

Prompt diagnosis is required to prevent bowel infarction. Contrast studies may show ascites, bowel thickening, or inflammation. Unfortunately, contrast can obscure the view of the arteries. The chest radiograph, unless it shows free air, is not helpful. The bowel pattern on abdominal films usually shows ileus, ascites, or a gasless abdomen.

Gas may handicap ultrasound. Lower endoscopy is not helpful because the distal colon is preserved. With a SMA embolic ischemia, the SMA embolus affects the small bowel and proximal colon (based on blood supply). An extended peritoneal lavage that reveals bloody fluid or an ischemia bowel can also be a helpful bedside test.

#### **REFERENCES (6)**

Bakal CW, Sprayregen S, Wolf EL. Radiology in intestinal ischemia: angiographic diagnosis and management. Surg Clin North Am 1992;72:125-141.

Boley SJ, Feinstein FR, Sammartano R, et al. New concepts in the management of emboli of the superior mesenteric artery. SGO 1981;153:561-569.

Boley SJ, Sprayregan S, Siegelman SS, et al. Initial results from an aggressive roentgenological and surgical approach to mesenteric ischemia. *Surgery* 1977; 82:848-855.

Falkensammer J, Oldenburg WA. Surgical and medical management of mesenteric ischemia. Curr Treat Options Cardiovasc Med 2006;8:137-143.

Flinn WR, Rizzo RJ, Park JS, et al. Duplex scanning for assessment of mesenteric ischemia. Surg Clin North Am 1990;70:99-107.

Kaleya RN, Boley SJ. Acute mesenteric ischemia. Crit Care Clin 1995;11:479-512.

Levy AD. Mesenteric ischemia. Radiol Clin North Am 2007;45:593-599.

Valverde FM, Pina FM, Aguado MM, et al. Gastrointestinal: Acute mesenteric ischemia. J Gastroenterol Hepatol 2005;20:1457.

RATIONALE (7)

Answer: C

Patients with acute pancreatitis may require intensive monitoring and treatment, given the potential for development of progressive organ dysfunction and/or life threatening complications. Decisions regarding admission of patients with acute pancreatitis to the ICU may be difficult in the initial stages of disease, secondary to a lack of strong predictors of illness at this phase. Traditionally, disease-specific scoring systems, such as the Ranson criteria and the Glasgow coma scale score have been utilized to identify patients at high risk of adverse outcomes. However, these criteria usually evaluate changes both in clinical and laboratory findings over 48 hours, and may not be as useful in the initial evaluation of a patient for triage decisions.

Of the above answers, the correct answer is C, an Acute Physiology and Chronic Health Evaluation (APACHE) II score of 12. Published reports of patients with acute pancreatitis have demonstrated that generic severity disease scoring systems, such as the acute physiology and chronic health evaluation II (APACHE II), were superior to disease specific scoring systems in predicting mortality. An APACHE II score >8, even at hospital admission, is predictive of a severe attack and can be used as a tool to make triage decisions.

Abdominal guarding is not a good triage criterion, as it is common even in less severe cases of acute pancreatitis. As long as other abdominal catastrophes, such as a perforated viscus, have been excluded, this sign does not correlate with either severity or likelihood of complications. Neither serum amylase or lipase levels correlate with severity of disease and should not be utilized for triage decisions.

A CT scan of the abdominal is a useful tool in assessing the severity of disease when it demonstrates necrosis of the pancreas; the degree of necrosis will correlate with overall severity of illness. However, in over 50% of the patients, as least 96 hours are needed for necrotic areas to be demonstrated on CT scans. Therefore, a normal CT scan in the initial 24 hours is not useful in predicting severity of complications.

Finally, while a threefold increased in aspartate aminotransferase or alanine amino transferase are both helpful in identifying gallstones as the cause of acute pancreatitis, they do not correlate with the severity of the disease or predict potential development of complications.

#### **REFERENCES (7)**

Besselink MG, van Santvoort HC, Witteman BJ, Gooszen HG. Management of severe acute pancreatitis: It's all about timing. *Curr Opin Crit Care* 2007;13:200-206.

Nathens AB, Curtis JR, Beale RJ, et al. Management of the critically ill patient with severe acute pancreatitis. *Crit Care* Med 2004;32:2524-2536.

Papachristou GI, Clermont G, Sharma A, et al. Risk and markers of severe acute pancreatitis. Gastroenterol Clin North Am 2007;36:277-296.

Tenner S. Initial management of acute pancreatitis: critical issues during the first 72 hours. Am J Gastroenterol 2004;99:2489-2494.

Swaroop VS, Chari ST, Clain JE. Severe acute pancreatitis. JAMA 2004;291:2865-2868.

RATIONALE (8)

Answer: C

This patient presents with a clinical picture consistent with acute pancreatitis, most likely secondary to alcohol abuse. The CT scan shown in the question demonstrates a swelling of the pancreas with areas of necrosis. Necrosis on the CT scan is an important marker of severity.

Given the options listed, the most appropriate management for this patient is total enteral nutrition via naso-jejunal feeding tube. The use of early enteral nutrition via naso-jejunal feeding tubes for patients with acute necrotizing pancreatitis seems to be associated with fewer complications and improved outcomes, as compared to patients who received parenteral nutrition.

Over the last years, the treatment of acute necrotizing pancreatitis has changed. Several studies and case series have demonstrated improved outcomes with medical management, delaying surgical interventions. Emergent laparotomy is not indicated as this point. If there is evidence of infection (which usually does not develop until later in the course of the disease), surgery should be considered. Other indications for surgery would include perforated viscus or other abdominal catastrophes.

The use of antibiotics in the treatment of acute necrotizing pancreatitis has created some controversy over the last several years. It is clearly indicated to utilize antibiotics when there is a documented infection of the pancreas or pseudocyst. Again, these infections are uncommon in the first several days of acute pancreatitis and are more prevalent after a week. When there is suspicion of infection, sampling of the pancreatic fluid collection or tissue should be done, preferably by percutaneous needle aspiration.

The institution of an empiric prophylactic has been studied in several small trials. Although some studies have suggested an advantage of using imipenem in patients who present with CT scan findings similar to this patient, the use of prophylactic-empiric antibiotics is not currently recommended in early acute pancreatitis without early documented infection.

Finally, there is no role for systemic corticosteroid therapy in patients with acute necrotizing pancreatitis.

#### REFERENCES (8)

Besselink MG, van Santvoort HC, Witteman BJ, Gooszen HG. Management of severe acute pancreatitis: It's all about timing. *Curr Opin Crit Care* 2007;13:200-206.

Kyriakidis AV, Raitsiou B, Sakagianni A, et al. Management of acute severe hyperlipidemic pancreatitis. *Digestion* 2006;73:259-264.

Nathens AB, Curtis JR, Beale RJ, et al. Management of the critically ill patient with severe acute pancreatitis. *Crit Care Med* 2004;32:2524-2536.

Tenner S. Initial management of acute pancreatitis: critical issues during the first 72 hours. Am J Gastroenterol 2004; 99:2489-2494.

Swaroop VS, Chari ST, Clain JE. Severe acute pancreatitis. JAMA 2004;291:2865-2868.

Werner J, Feuerbach S, Uhl W, Büchler MW. Management of acute pancreatitis: from surgery to interventional intensive care. *Gut* 2005;54:426-436.

RATIONALE (9)

Answer: C

This patient presents with a clinical picture that is consistent with fulminant liver failure (FLF). Acute liver failure (ALF) is defined as a rapid deterioration of hepatic function, manifested by an increase in prothrombin time (PT) and a decrease of factor V, without evidence of hepatic encephalopathy. Fulminant hepatic failure is a severe acute liver failure complicated by hepatic encephalopathy in a patient with no previous liver disease. Usually the interval between the onset of liver failure is manifested by jaundice. The onset of hepatic encephalopathy is less than two weeks.

The most common cause of ALF and fulminant hepatic failure (FHF) in the United States is acetaminophen toxicity. Complications related to, ALF and FHF include coagulopathy, cerebral edema and intracranial hypertension, acute portal hypertension, renal failure, infections, and multiple organ failure. The most common cause of death in patients with FHF is cerebral edema, secondary to increased intracranial pressure, leading to herniation.

The care for patients with FHF and ACF is supportive. It is important to identify patients at higher risk of death for rapid referral to transplant centers, since in most cases, liver transplant is the only curative therapeutic modality.

#### **REFERENCES (9)**

Ganger D. Liver failure. In Dellinger P, Parrillo J, eds. *Critical Care Medicine* 2nd ed. Philadelphia, PA: Mosby; 2001.

Jalan R. Acute liver failure: current management and future prospects. *J Hepatol* 2005;42:S115-S223. O'Grady JG. Acute liver failure. *Postgrad Med J* 2005;81:148-54.

RATIONALE (10)

Answer: B

Recognized markers of the risk of severe acute pancreatitis include specific laboratory values that measure the systemic inflammatory response such as the C-reaction protein. Scoring systems that asses inflammation or organ failure, such as the ransom score used in imaging studies, are also employed. Up to 57% of patients who are hospitalized with acute pancreatitis will have fluid collections, with 39% having 2 areas involved, and 33% having 3 or more. Fluid collections with very high levels of pancreatic enzymes are usually associated with pancreatic duct disruptions and may eventually form pseudocysts.

An important complication that can develop during the first few days of pancreatitis is pancreatic necrosis. Pancreatic necrosis occurs as diffuse or focal areas of non-viable pancreatic parenchymal, and is best demonstrated by the loss of tissue perfusion on contrast-enhanced CT scans. Infection of necrotic tissue is suspected when there is a fever leukocytosis with a failure to improve, or an unexpected deterioration, usually after the first week of illness. The diagnosis of an infected necrosis is usually made by a fine needle aspirate of the necrotic area, guided by either a CT scan or an ultrasound, with Gram staining and culture of the aspirate.

Ensuring adequate nutrition is important in patients with severe complicated pancreatitis. Enteral feeding is often successful and may actually be superior to total parenteral nutrition when tolerated. It is usually well tolerated in the patient's ileus.

Surgical intervention is indicated in patients with infected pancreatic necrosis. However, surgery within the first few days after the onset of severe acute pancreatitis is associated with death rates of up to 65%. Furthermore, there is no clear demarcation between viable and nonviable tissue early in the course of acute pancreatitis. Delaying surgical debridement of necrotic tissue for at least two weeks, if possible, while the patient's medical condition is optimized and viable pancreatic tissue becomes evident, has been supported by observational data.

#### **REFERENCES (10)**

- Al-Omran M, Groof A, Wilke D. Enteral versus parenteral nutrition for acute pancreatitis. *Cochrane Database Syst* Rev 2003;1:CD002837.
- Arvanitakis M, Delhaye M, De Maertelaere V, et al. Computed tomography and magnetic resonance imaging in the assessment of acute pancreatis. *Gastroenterology* 2004;126:715-723.
- Bassi C, Larvin M, Villatoro E. Antibiotic therapy for prophylaxis against infection of pancreatic necrosis in acute pancreatitis. *Cochrane Database Syst* Rev 2003;4:CD002941.
- Meier R, Beglinger C, Layer P, et al. ESPEN guidelines on nutrition in acute pancreatitis: European Society of Parenteral and Enteral Nutrition. *Clin Nutr* 2002;21:173-183.
- Nathens AB, Curtis JR, Beale RJ, et al. Management of the critically ill patient with severe acute pancreatitis. *Crit Care Med* 2004;32:2524-2536.
- Ranson JH. Etiological and prognostic factors in human acute pancreatitis: a review. Am J Gastroenterol 1982;77:633-638.
- Rau B, Pralle U, Mayer JM, Beger HG. Role of ultrasonographically guided fine-needle aspiration cytology in the diagnosis of infected pancreatic necrosis. *Br J Surg* 1998;85:179-184.
- Pederzoli P, Bassi C, Vesentini S, Campedelli A. A randomized multicenter clinical trial of antibiotic prophylaxis of septic complications in acute necrotizing pancreatitis with imipenem. *Surg Gynecol Obstet* 1993;176:480-483.
- Whitcomb DC. Acute pancreatitis. N Engl J Med 2006;354:2142-2150.

RATIONALE (11)

Answer: C

Approximately 10% of all cases of acute cholecystitis are acute acalculous cholecystitis (AAC). The precise mechanisms are unknown, but the most common theories of pathogenesis relate to bowel stasis, sepsis, and ischemia.

The majority of patients who develop AAC are outpatients. The age of onset of patients with AAC is known to be older than patients with calculous cholecystitis. Although calculous gallbladder disease is more common in females, AAC is more common in males (2.1:1). AAC may also occur from a secondary infection of the gallbladder following systemic infection by either bacteria or fungi.

Sonography and CT scanning are both highly sensitive and specific, whereas hepatobiliary scanning offers frequent false positives. CT scanning better defines presence and degree of pericholecystic inflammation.

#### **REFERENCES (11)**

Batey A, Khan MA. Acalculous cholecystitis. Clin Gastroenterol Hepatol 2007;5:e8.

Fox MS, Wilk PJ, Weissmann HS, et al. Acute acalculous cholecystitis. *Surg Gynecol Obstet* 1984;159:13-16.

Haitt JR, Kobayashi JR, Doty JE, et al. Acalculous candida cholecystitis: a complication of critical surgical illness. *Am Surg* 1992;57:835-829.

Owen CC, Jain R. Acute Acalculous Cholecystitis. *Curr Treat Options Gastroenterol* 2005;8:99-104. Ryu JK, Ryu KH, Kim KH. Clinical features of acute acalculous cholecystitis. *J Clin Gastroenterol* 2003;36:166-169.

Savoca PE, Longo WE, Zucker KA, et al. The increasing prevalence of acalculous cholecystitis in outpatients. Results of a 7-year study. *Ann Surg* 1990;211:433-437

RATIONALE (12)

Answer: A

Charcot described the triad of pain, fever, and jaundice due to acute obstructive cholangitis in 1877. However, Boey and Way (1) report the triad to be incomplete in 30% of cases. In their series, acute cholangitis resulted predominately from benign postoperative biliary stricture and common duct stones (36 and 30%, respectively), less frequently from pancreastic or biliary duct neoplasm, with the remaining cases falling into the miscellaneous category. Beinhart and colleagues (2) reported that of 150 jaundiced patients with documented biliary obstruction who were studied by transhepatic cholangiography, 10.7% had no evidence of biliary duct dilation despite bilirubin levels ranging from 1.5 to 27.1 mmol/dL.

In a comprehensive review of the literature, Sievert and Vaki (3) suggested that hepatobiliary scintigraphy is the most reliable for making the diagnosis of biliary obstruction, whereas ultrasonography either failed to visualize common duct stones or intrahepactic bile duct dilation. Operative mortality for acute cholangitis ranges from 17 to 40% with worsening mortality associated with age and concurrent illnesses.

Boey and Way reported that 85% of their patients who presented with acute cholangitis with moderate signs and symptoms could be managed initially with IV fluids, careful monitoring, and broad-spectrum antibiotics with great success. Any worsening of symptoms or failure to improve rapidly required surgical exploration. Of the 99 patients evaluated, 15 patients required immediate surgical drainage. Their operative mortality rate was 40%. Of the 84 remaining patients managed nonoperatively, only 1 died.

If the initial management of the patient can be accomplished nonoperatively, authors disagree as to the definitive management. Pessa and associates (4) suggest that acute drainage of the biliary tree via transhepatic catheterization should be performed before definitive internal drainage procedures. In their series of 42 patients managed with transhepatic drainage catheters (17% of whom failed to demonstrate dilated intrahepatic ducts), all patients showed signs of resolution of sepsis within 24 hours of the procedure. The complication rate of the procedure itself was 7%, with the overall mortality rate being 5%.

By contrast, other authors report success in relieving sepsis through the performance of a definitive drainage procedure with an endoscopic sphincterotomy. Press et al. reported on 71 patients who failed initial medical therapy. Thirty-nine percent of these patients underwent surgical decompression, while 61% received endoscopic sphincterotomy. The morbidity and mortality rates for the surgical group were 57 and 21.4%, respectively, while the morbidity and mortality rates for endoscopic sphincterotomy were 28 and 4.7%, respectively. There was no difference in the incidence of retained stones from either approach.

#### **REFERENCES (12)**

Bornman PC, van Beljon JI, Krige JE. Management of cholangitis. *J Hepatobiliary Pancreat Surg* 2003;10:406-414.

Yusoff IF, Barkun JS, Barkun AN. Diagnosis and management of cholecystitis and cholangitis. Gastroenterol Clin North Am 2003;32:1145-1168.

# SECTION 5: Hematology/Oncology

### **SECTION 5: HEMATOLOGY/ONCOLOGY**

Instructions: For each question, select the most correct answer.

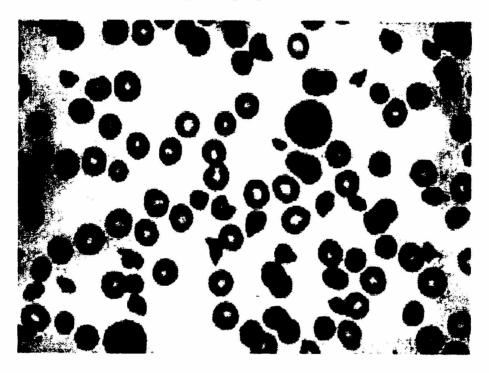
1. A 50-year-old chronic dialysis patient is admitted to the ICU at 11:00 pm with altered mental status for 2 days, fever, tachycardia, and hypoxemia. A chest radiograph reveals a right lower lobe infiltrate. Prolonged bleeding from venipuncture sites and bleeding from the nasal mucosa after passage of a nasogastric tube is also noted. Platelet count is 100,000/mm³, prothrombin time 11.5 seconds, partial thromboplastin time 35 seconds, blood urea nitrogen 150 mg/dL, and creatinine 10 mg/dL.

Which one of the following is most likely to result in rapid improvement in the bleeding diathesis?

- A. Platelet transfusion
- B. Hemodialysis for uremia
- C. Conjugated estrogens
- D. Cryoprecipitate
- E. Desmopressin acetate

2. A 55-year-old male with coronary artery disease and hypertension is transferred from the telemetry unit to the ICU after developing an altered mental status and fever. The patient underwent percutaneous coronary angioplasty 1 month ago with a stent placement and was recently admitted to the hospital for the evaluation of chest pains. On physical examination, temperature is 39.9°C (103.9°F), HR 115/min, RR 25/min and BP 120/90 mm Hg. There is no jugular vein distention, no S3, lungs are clear on auscultation, the abdominal examination is unremarkable and extremities are cool and mottled. Neurologic examination reveals a confused patient who is able to move all extremities spontaneously and opens his eyes to voice.

Laboratory data include hemoglobin 8.1 g/dL, white blood cells 10,000/mm<sup>3</sup>, platelet count 4,000/mm<sup>3</sup>, partial thromboplastin time 25 seconds, international normalized ratio 1.0, prothrombin time 12 seconds, sodium 139 mEq/L, potassium 4.5 mEq/L, chloride 102 mEq/L, bicarbonate 22 mEq/L, and serum creatinine 2.2 mg/dL. A peripheral smear of the blood is shown in the figure below.



Which one of the following is most appropriate as initial treatment for this patient?

- A. Argatroban
- B. Dexamethasone
- C. Plasmapheresis
- D. Low-molecular-weight heparin
- E. IV gamma globulin

3. A 40-year-old woman is admitted to the hospital with probable immune thrombocytopenic purpura (ITP). She has some petechiae on her lower extremities and ecchymosis in areas of mild trauma. Her platelet count is 5,000/mm<sup>3</sup> and prothrombin time/partial thromboplastin time are normal. She is started on prednisone at 80 mg/day. On day 2 in the hospital, she slipped and fell, resulting in a femoral neck fracture. Surgical repair is required in the next several days.

Which one of the following is the most appropriate intervention to increase the platelet count prior to surgery?

- A. Platelet transfusion
- B. Splenectomy
- C. IV immunoglobulin
- D. Rituximab
- E. Danazol

4. A 65-year-old female is admitted to the ICU after bypass surgery for acute myocardial infarction with cardiogenic shock. The postoperative course is complicated by acute renal insufficiency, and on the postoperative day 5, the patient develops acute dyspnea with hypoxemia. A diagnosis of left lower extremity deep venous thrombosis is made by lower extremity ultrasound, with probable pulmonary embolism. The patient is started on IV unfractionated heparin (weight-based nomogram) to achieve a partial thromboplastin time 1.5 to 2 times the normal value.

Laboratory studies at this time reveal normal electrolytes, a blood urea nitrogen 45 mg/dL, creatinine 2.8 mg/dL, WBC count 9,000/mm³, hemoglobin 10.1 g/dL, platelets 250,000/mm³, normal prothrombin time, international normalized ratio, and partial thromboplastin time. After 2 days of therapeutic anticoagulation with unfractionated heparin, the patient complains of right lower extremity pain. Examination of the right lower extremity reveals a cold painful extremity with loss of distal pulses. Laboratory studies are significant for a blood urea nitrogen of 50 mg/dL, creatinine 2.8 mg/dL, platelets 90,000 /mm³, an international normalized ratio of 1.2, and partial thromboplastin time 50 seconds.

Which one of the following therapeutic options is most appropriate at this time?

- A. Increase unfractionated heparin to achieve a higher partial thromboplastin time
- B. Discontinue unfractionated heparin, and start enoxaparin
- C. Discontinue unfractionated heparin, and start warfarin
- D. Discontinue unfractionated heparin, and start argatroban
- E. Discontinue anticoagulation

5. A 22-year-old male with a gun shot wound to the chest is returned to the ICU postoperatively and is noted to be oozing blood from the incision site, chest tube site, and recent venipunctures. He received 4 U of packed red blood cells and 1,000 mL of cell saver blood intraoperatively. There was no surgical bleeding prior to closure and the postoperative hemoglobin was 9.6 g/dL. Vital signs include BP 100/60 mm Hg, HR 80/min, RR 14/min (on ventilator), temperature 32.2°C (90°F). Coagulation studies reveal a platelet count of 100,000/mm³, prothrombin time 14.2 seconds, partial thromboplastin time 40.0 seconds, and D-dimer negative.

Which one of the following is the most appropriate intervention for the bleeding?

- A. Transfuse platelets
- B. Warm the patient
- C. Transfuse cryoprecipitate
- D. Transfuse fresh frozen plasma
- E. Observe the patient for any further clinical changes
- 6. A 22-year-old otherwise healthy male was involved in a motor vehicle accident and suffered blunt abdominal trauma. After exploratory laparotomy with a splenectomy, he required transfusion of 4 U of packed red blood cells. Postoperatively, he had diffuse oozing at the site of the incision. His laboratory studies preoperatively revealed a platelet count 210,000/mm³, prothrombin time of 12.8 seconds, and partial thromboplastin time of 48 seconds.

Which one of the following is the most likely etiology of this patient's bleeding?

- A. Disseminated intravascular coagulation
- B. Hypothermia
- C. von Willebrand disease
- D. Salicylate use
- E. Citrate toxicity

7. A patient with septic shock secondary to pneumonia was admitted and treated in the ICU. On the hospital day 3, the patient's BP is stable, and he is being weaned from norepinephrine. The patient's hemoglobin is 8.5 g/dL, platelets 35,000/mm³, prothrombin time 18 seconds, and partial thromboplastin time 40 seconds.

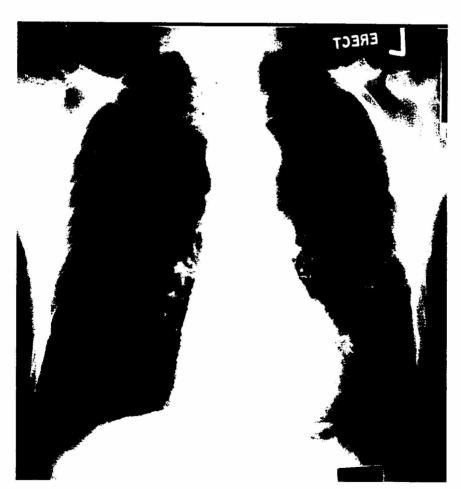
Which blood products should be transfused at this time?

- A. Red blood cells, platelets, and fresh-frozen plasma
- B. Red blood cells and platelets
- C. Red blood cells
- D. Platelets
- E. No blood products
- 8. Which one of the following is most correct concerning fresh frozen plasma (FFP)?
  - A. Rapid infusion increases the frequency of febrile and urticarial reactions
  - B. FFP may be used in patients with antithrombin deficiency when other preparations are unavailable
  - C. Platelet transfusion increases the requirement for FFP
  - D. FFP is an efficient source of fibrinogen
  - E. Continuous infusion achieves adequate factor levels for hemostasis

9. A 68-year-old male with a 100 pack/year smoking history is admitted to the ICU with hypotension. He has a 2-month history of weight loss and fatigue and 3-day history of anorexia and polyuria. On admission, his temperature is 37°C (98.6°F), BP 90/50 mm Hg, HR 114/min, and RR 18/min. The patient is lethargic but has no focal neurologic deficits. Initial laboratory examinations revealed sodium 148 mmol/L, potassium 4.2 mmol/L, chloride 110 mmol/L, CO<sub>2</sub> 28 mmol/L, blood urea nitrogen 35 mg/dL, creatinine 2.1 mg/dL, and glucose 120 mg/dL. ECG reveals sinus tachycardia and shortened QT interval. The admission chest radiograph is shown below.

In addition to IV normal saline solution, which one of the following will most likely benefit this patient?

- A. Methylprednisolone
- B. Magnesium sulfate
- C. Pamidronate
- D. Sodium phosphate
- E. Mannitol



10. A 28-year-old male with hemoglobin SC disease was admitted to the hospital for leg and chest pain secondary to a vasoocclusive crisis. On admission, the chest radiograph showed mild cardiomegaly but no infiltrates, and the room air pulse oximetry saturation was 97%. He was treated with IV hydration and analgesics.

On hospital day 3, the patient developed a fever of 38°C (100.4°F) and shortness of breath. He continued to complain of sternal pain. The oxygen saturation on room temperature was 85% and rales were noted in the right lower lobe. The chest radiograph confirmed infiltrates in the right and left lower lobe. His hemoglobin at this time was 9.2 g/dL, with a white blood cell count of 20,000/mm³ (unchanged from admission). The patient is transferred to the ICU for further care.

Which of the following interventions is most appropriate for this patient?

- A. Exchange transfusion
- B. Simple transfusion
- C. Administration of IV dexamethasone
- D. Bronchoalveolar lavage to exclude infection
- E. Administration of inhaled nitric oxide
- 11. A 20-year-old with hemophilia A sustained significant chest wall trauma in a motor vehicle accident. After thoracostomy for a right pneumothorax, he has 100 mL/h of bloody output from the chest tube. He has been transfused multiple times in the past and is known to have inhibitors to factor VIII.

Which one of the following should be administered?

- A. Recombinant factor VIII
- B. Cryoprecipitate
- C. Factor IX concentrate
- D. Recombinant factor VIIa
- E. Fresh frozen plasma

12. A 65-year-old male with chronic lymphocytic leukemia was initiated on outpatient infusion pump therapy with fludarabine at a daily dose of 25 mg/m<sup>2</sup>. His hemoglobin was 11.4 g/dL, white blood cell count 90,000/mm<sup>3</sup> with 95% lymphocytes, platelets 108,000/mm<sup>3</sup>, creatinine 1.0 mg/dL, blood urea nitrogen 23 mg/dL, uric acid 7 mg/dL, and lactic dehydrogenase 597 U/L.

At the initiation of treatment, he had massive splenomegaly and bulky intraabdominal and retroperitoneal lymphadenopathy. He presented to the hospital on the 4th day of treatment complaining of flank pain, weakness and nausea. His serum creatinine was 2.4 mg/dL, blood urea nitrogen 55 mg/dL, uric acid 18 mg/dL, and lactic dehydrogenase 1,800 U/L. His potassium was 5.9 mmol/L.

In addition to IV fluids, which one of the following interventions should be initiated?

- A. Allopurinol
- B. Immediate hemodialysis
- C. Immediate continuous venovenous hemofiltration /dialysis
- D. Urine acidification
- E. IV furosemide
- 13. A 50-year-old man underwent coronary artery bypass graft surgery, with a transfusion of 3 U of packed red cells intraoperatively. He was extubated in the postoperative care unit, but developed tachypnea and shortness of breath 2 hours later. His ECG was unchanged, and he denied any chest pain. His hemoglobin was 10.9 g/dL and electrolytes were within the normal range. The chest tube had minimal bloody drainage. A pulmonary artery catheter was inserted, with findings of a pulmonary artery occlusion pressure 12 mm Hg and cardiac index of 3.2 L/min/m². His blood gas on a nonrebreather mask was pH 7.45, Paco<sub>2</sub> 34 mm Hg, and Pao<sub>2</sub> 80 mm Hg. His chest radiograph showed diffuse bilateral infiltrates.

Which one of the following is the most appropriate intervention?

- A. Anticoagulate with heparin
- B. Administer corticosteroids
- C. Administer diuretics
- D. Administer antibiotics
- E. Continue respiratory support

14. A 50-year-old man was admitted to the hospital for evaluation of headache, weakness, weight loss and anemia of 1 month duration. His laboratory results were hemoglobin 8.2 g/dL, hematocrit 25%, white blood cell count 8.2 x 10³/mm³, platelets 160,000/mm³, normal electrolytes, blood urea nitrogen 35 mg/dL, creatinine 2.2 mg/dL, AST 60 U/L, ALT 32 U/L, total bilirubin 1.2 mg/dL, total protein 9.8 mg/dL, and albumin 2.2 mg/dL. The physical examination revealed mild hepatosplenomegaly and pallor. On the second hospital day, the patient was found to be lethargic and confused with decreased movement of his right arm and leg. The patient was transferred to the ICU for further care.

Which one of the following interventions is most appropriate?

- A. Thrombolytics after a negative head CT scan
- B. Plasmapheresis
- C. Transfusion of 2 U red blood cells
- D. Corticosteroids
- 15. A 40-year-old male with a recent diagnosis of small-cell lung cancer presents to the hospital with a history of fever for 2 days. The patient has recently completed a course of chemotherapy 1 day ago. The patient denies cough, dysuria, or diarrhea. On examination, vital signs are temperature 39.4°C (103°F), HR 105/min, RR 16/min, and BP 100/60 mm Hg. The patient is neutropenic. The patient has a right subclavian porta catheter, but the site is nontender and without erythema. Lungs are clear, and the rest of the examination is unremarkable.

Laboratory data are significant for a hemaglobin of 10 g/dL, white blood cell count of  $400/\mu L$ , and a platelet count of 80,000/mL. A chest radiograph is obtained and shows no infiltrates.

Which one of the following is the most appropriate antibiotic regimen for the patient?

- A. No antibiotics pending results of blood cultures
- B. Ceftazidime
- C. Ceftazidime + vancomycin
- D. Ceftazidime + vancomycin + fluconazole
- E. Ceftazidime + amikacin

### **SECTION 5: HEMATOLOGY/ONCOLOGY**

#### **ANSWERS:**

1-E; 2-C; 3-C; 4-D; 5-B; 6-C; 7-E; 8-B; 9-C; 10-A; 11-D; 12-A; 13-E; 14-B; 15-B

RATIONALE (1)

Answer: E

Uremia is one of the most common causes of platelet dysfunction in the critically ill, and bleeding from invasive procedures and surgery may be difficult to control. Several options exist to correct platelet dysfunction. Hemodialysis to lower the blood urea nitrogen would be optimal but usually requires considerable time to institute at night in many situations. Hemodialysis may also contribute to bleeding by continuous platelet activation, induced by the interaction between blood and artificial surfaces, resulting in platelet exhaustion. Continuous renal replacement techniques would not be expected to improve the uremia rapidly.

Desmopressin acetate (DDAVP) administered intravenously at a dose of 0.3 µg/kg has a rapid effect on improving platelet function. The onset of action is within 1 hour and the effects last 4-8 hours. Subcutaneous injection and nasal spray are alternative routes of administration. DDAVP results in a 2- to 6-fold increase in both factor VIII and von Willebrand factor, which increases platelet adhesiveness. Unfortunately, this effect is short-lived and patients rapidly develop tachyphylaxis. DDAVP is most helpful in controlling emergent bleeding while other measures are being instituted.

Platelet transfusions would be of no benefit since transfused platelets are exposed to the same uremic environment. Conjugated estrogens are of value in improving platelet dysfunction, but the effect takes several days. Therefore, they are not an option for emergent interventions. Erythropoietin is another long-term treatment option that improves platelet function.

Cryoprecipitate has been used in the past, but is no longer recommended because it is not effective in many patients. It also exposes the patient to transfusion-related complications. Keep in mind that the use of antibiotics such as penicillins and certain cephalosporins may also potentiate platelet dysfunction.

#### REFERENCES (1)

Boccardo P, Remuzzi G, Galbusera M. Platelet dysfunction in renal failure. Semin Thromb Hemostasis 2004;30:579-589.

Carvalho AC. Acquired platelet dysfunction in patients with uremia. *Heme Oncol Clin N Amer* 1990;4:129-143.

DeLoughery TG. Management of bleeding with uremia and liver disease. *Curr Opin Hematol* 1999;6:329-633.

RATIONALE (2)

Answer: C

This patient has thrombotic thrombocytopenic purpura (TTP), most likely as a result of treatment with ticlopidine after his cardiac catheterization. TTP is characterized by the pentad of thrombocytopenia, microangiopathic hemolytic anemia, fever, neurologic findings, and renal dysfunction. These clinical features rarely present simultaneously. The peripheral smear that is shown has fragmented red cells and schistocytes, which are typical findings for microangiopathic hemolytic anemia. In the context of a clinical presentation, TTP is the most likely diagnosis. Patients do not require all elements of the pentad to be fulfilled for diagnosis of TTP, and a high level of vigilance must be maintained to identify these patients in a timely fashion and institute appropriate treatment. TTP is associated with invasive enteric infections, chemotherapeutic agents such as mitomycin C, cancer, HIV infection, and anti-platelet agents (ticlopidine and clopidogrel). There is also an idiopathic form of TTP. TTP seems to be initiated by endothelial damage that leads to the accumulation of large von Willebrand factor multimers that lead to platelet aggregation and thrombin formation in affected organ systems.

The treatment of TTP, regardless of the cause, is the discontinuation of the offending agent and daily plasma exchange (plasmapheresis plus infusion of fresh frozen plasma). Prompt therapy with plasma exchange decreases the mortality of TTP to 10%, and permanent organ dysfunction is uncommon. In severe cases refractory to conventional therapy, glucocorticoid therapy, and also splenectomy, have been reported to be useful adjunctive measures.

Patients with TTP in general should not be transfused with platelets (unless there is life-threatening or intracranial bleeding). Argatroban is reserved for patients who develop heparin-induced thrombocytopenia and require systemic anticoagulation. There has been no proven benefit or role for systemic corticosteroids in the initial treatment of TTP. Low-molecular weight heparin and immunoglobulin are not indicated in TTP.

#### **REFERENCES (2)**

- George JN. Thrombotic thrombocytopenic purpura. *N Engl J Med* 2006;354:1927-1935. Guidelines on the diagnosis and management of the thrombotic microangiopathic haemolytic anaemias. *Br J Haematology* 2003;120:556-573.
- Kwaan HC, Boggio LN. The clinical spectrum of thrombotic thrombocytopenic purpura. Semin Thromb Hemostasis 2005;31:673-680.
- Moake JL. Thrombotic microangiopathies. N Engl J Med 2002;347:589-600.
- Medina PJ, Sipols JM, George JM. Drug-associated thrombotic thrombocytopenic purpurahemolytic uremic syndrome. *Curr Opin Hematol* 2001; 8:286-293.
- Rock G. The management of thrombotic thrombocytopenic purpura in 2005. Semin Thromb Hemostasis 2005;31:709-716.

RATIONALE (3)

Answer: C

Patients with immune thrombocytopenic purpura (ITP) usually do not have significant bleeding because the few platelets present are young and active in coagulation. This patient has a comorbidity that makes urgent surgical intervention necessary. Although steroids are the first line treatment, platelet counts may not increase for several days to weeks. Intravenous immunoglobulin would be the best intervention to accomplish a transient increase in platelets during the operative intervention. It is administered as 0.5-1 g/kg/day, along with methylprednisolone 1 g/ day. Depending on the response, platelet transfusions may be needed at the time of surgery.

Another option is the use of anti-D immunoglobulin, but this is an option only in patients who are Rh D positive and have a spleen. The hemolysis that can occur with anti-D immunoglobulin would also preclude its use in a patient undergoing surgery.

Platelet transfusions alone are unlikely to benefit this patient, since they would be subject to the same antibody destruction as endogenous platelets. Splenectomy would not be warranted; rituximab and danazol are options for chronic ITP and would be unlikely to have rapid effects on the platelet count.

#### **REFERENCES (3)**

Cines DB, Blanchette VS. Immune thrombocytopenic purpura. *N Engl J Med* 2002;346:995-1008. Stasi R, Provan D. Management of immune thrombocytopenic purpura in adults. *Mayo Clin Proc* 2004;79:504-522.

RATIONALE (4)

Answer: D

This patient has developed heparin-induced thrombocytopenia (HIT) type II with arterial thrombosis of the right lower extremity. There are 2 types of heparin-associated thrombocytopenia. Type I occurs in 10 to 20% of patients receiving unfractionated heparin. Nonimmune factors result in mild decreases in platelet counts after 1 to 4 days of therapy with heparin. Most patients will normalize the platelet count in a few days despite heparin continuation. Type II is more severe and is associated with thrombotic complications in 30 to 80% of cases. Development of type II HIT is related to the formation of heparin-platelet factor 4 complexes and the induction of immunoglobulin G antiheparin-PF4 antibodies. Type II HIT usually develops after 5 days of heparin exposure. However, in patients with prior exposure to heparin it may appear earlier. HIT may also occur after heparin is discontinued.

Cardiac and orthopedic surgery patients and ICU patients are at higher risk of developing HIT. Once type II HIT is suspected, all sources of heparin, including low-molecular weight heparins, should be discontinued. Patients with thrombotic complications (venous or arterial) from HIT, and/or other indications for anticoagulation should be treated with antithrombin agents such as lepirudin or argatroban, or danaparoid (not available in the United States).

However, lepirudin has renal elimination and needs dose adjustment for renal dysfunction. Therefore, in patients with renal insufficiency, argatroban would be the preferred choice, since it is hepatically-metabolized.

Warfarin anticoagulation can exacerbate the prothrombotic state of type II HIT prior to achieving full anticoagulation with an international normalized ratio >2.0. As a consequence, warfarin administration should be withheld until there is evidence of improvement and the patient is fully anticoagulated with a parenteral antithrombin agent.

#### **REFERENCES (4)**

Arepally GM, Ortel TL. Heparin-induced thrombocytopenia. *N Engl J Med* 2006;355:809-817. Greinacher A, Farner B, Kroll H, et al. Clinical features of heparin-induced thrombocytopenia including risk factors for thrombosis. *Thromb Haemost* 2005;94:132-135.

Warkentin TE. Heparin-induced thrombocytopenia: pathogenesis and management. *Br J Haema* 2003;121:535-555.

Warkentin TE. Management of heparin-induced thrombocytopenia: a critical comparison of lepirudin and argatroban. *Thromb Res* 2003:110:73-82.

Warkentin TE, Greinacher A. Heparin-induced thrombocytopenia: recognition, treatment, and prevention: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* 2004;126(suppl):S311-S337.

RATIONALE (5)

Answer: B

In this patient, the most likely cause of the oozing is a dysfunction of platelets due to hypothermia. Clinical data should be reviewed to exclude uremia and medications as causes of platelet dysfunction. The treatment of choice is to warm the patient and reassess.

Postoperative bleeding can be due to multiple etiologies. The initial assessment of the patient should verify that vascular control was achieved at the time of surgery. Massive transfusion may result in dilution of platelets and coagulation factors leading to diffuse microvascular bleeding. This usually occurs when more than 1 blood volume is replaced within 12 to 24 hours. In this patient, the amount of blood transfused is unlikely to lead to this complication. In addition, the platelet count of 100,000/mm³ should be adequate for maintaining hemostasis, unless the platelets are dysfunctional. Therefore, transfusion of platelets is not warranted. The mildly prolonged prothrombin time and PTT would not be expected to result in significant bleeding unless other coagulation defects were present.

Transfusion of packed red blood cells may be appropriate in the setting of severe anemia, but would not correct a coagulopathy.

Cryoprecipitate is only indicated in the setting of severe depletion of fibrinogen.

#### **REFERENCES (5)**

Klein HG, Higgins MJ. Use of blood components in the intensive care unit. In Parrillo JE, Dellinger RP, eds. *Critical Care Medicine*. 2nd ed. St. Louis, MO: Mosby; 2001:1416-1438.

Redei I, Rubin RN. Recognizing the most common causes of bleeding in the ICU. *J Crit Illness* 1995;10:121-132.

Spahn DR, Rossaint R. Coagulopathy and blood component transfusion in trauma. *Br J Anaesthesia* 2005;95:130-139.

RATIONALE (6)

Answer: C

Von Willebrand disease is the most common inherited coagulation disorder. The most common type (type 1) is due to decreased release or production of von Willebrand factor (VWF). Other types are due to abnormal VWF polymers or decreased levels of VWF and VIII activity. Bleeding is usually only noted with trauma, surgery, and other invasive procedures. Impaired platelet adhesion at the vascular site of injury results in bleeding. Coagulation studies reveal only a prolonged partial thromboplastin time and increased bleeding time. Treatment of von Willebrand disease includes desmopressin acetate (DDAVP), factor VIII concentrate, and cryoprecipitate. DDAVP is effective in type 1 disease only. Factor VIII concentrate is preferred over cryoprecipitate due to the lower infection risk of the factor concentrate.

Acquired von Willebrand disease has also been described. Associated conditions include lymphoproliferative and myeloproliferative disorders and cardiovascular defects such as endocarditis and septal defects.

Salicylates would not prolong the partial thromboplastin time and citrate toxicity is unlikely since only 4 U of red blood cells were transfused. Hypothermia postoperatively can result in platelet dysfunction, but is unlikely to result in an isolated prolonged partial thromboplastin time. Likewise, the normal platelet count and normal prothrombin time make DIC an unlikely cause of the oozing.

#### **REFERENCES (6)**

Kumar S, Pruthi RK, Nichols WL. Acquired von Willebrand disease. *Mayo Clin Proc* 2002;77:181-187.

Mannucci PM. Treatment of von Willebrand's disease. N Engl J Med 2004;351:683-694.

RATIONALE (7)

Answer: E

In this stable patient recovering from septic shock without evidence of bleeding, no blood product is needed at this time. Based on the Canadian Critical Care Trials Group prospective study of transfusion thresholds, the transfusion of red blood cells is recommended only when hemoglobin decreases <7.0 g/dL, targeting a hemoglobin of 7.0 to 9.0 g/dL. The effect of red blood cell transfusion in septic patients has been evaluated in several studies and shows an increase in oxygen delivery but no increase in oxygen consumption. The transfusion threshold recommended in a stable patient is distinct from the target hematocrit of 30% in patients with low central venous oxygen saturation during the first 6 hours of resuscitation of septic shock.

No clinical trials have addressed the transfusion of platelets and fresh-frozen plasma (FFP) in septic patients, and recommendations are based on consensus opinion and experience in other patient groups. In patients with severe sepsis, platelets should be administered when counts are ≤5,000/mm³, regardless of apparent bleeding. Platelet transfusion can be considered when counts are 5,000 to 30,000/ mm³, and there is a significant risk for bleeding. Higher platelet counts may be required if surgery or invasive procedures are planned.

In the presence of active bleeding or prior to surgical or invasive procedures, FFP is indicated for coagulopathy due to a documented sufficiency of coagulation factors. In the absence of bleeding, routine use of FFP to correct laboratory abnormalities is not recommended.

#### REFERENCES (7)

- College of American Pathologists. Practice parameter for the use of fresh-frozen plasma, cryoprecipitate, and platelets, *JAMA* 1994;271;777-781.
- Hébert PC, Wells G, Blajchman MA, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. N Engl J Med 1999;340:409-417.
- Zimmerman JL. Use of blood products in sepsis: an evidence-based review. Crit Care Med 2004;32(suppl):S542-S547.

RATIONALE (8)

Answer: B

Fresh frozen plasma (FFP) includes all coagulation factors, including antithrombin. For patients with an antithrombin deficiency who experience bleeding or require surgery, FFP may be used to replace antithrombin when the commercial preparation is not available. Additional indications for transfusion of FFP include:

- coagulopathy due to a documented deficiency of coagulation factors with active bleeding or planned invasive procedures,
- massive blood transfusion with evidence of a coagulation deficiency, reversal of warfarin effect, and
- plasma exchange for thrombotic thrombocytopenic purpura or hemolytic uremic syndrome.

Rapid infusion is needed to achieve appropriate factor levels for hemostasis. The speed of infusion does not increase adverse reactions. Continuous infusion of FFP is inappropriate, since it does not usually result in adequate factor levels. FFP may need to be administered every 6 to 8 hours, since the half-life of factor VII is approximately 6 hours. Smaller doses of FFP may be needed if platelets are also transfused. The processing of platelets results in the presence of plasma, and for every 5 to 6 U random donor platelets, the patient may receive the equivalent of 1 bag of random donor FFP. FFP is not an efficient source of fibrinogen. For fibrinogen levels <100 mg/dL, cryoprecipitate is the best source of fibrinogen.

#### **REFERENCES (8)**

British Committee for Standards in Haematology, Blood Transfusion Task Force. Guidelines for the use of fresh-frozen plasma, cryoprecipitate and cryosupernatant. *Br J Haematology* 2004;126:11-18. College of American Pathologists. Practice parameter for the use of fresh-frozen plasma, cryoprecipitate, and platelets. *JAMA* 1994; 271:777-781.

Hellstern P, Muntean W, Schramm W, et al. Practical guidelines for the clinical use of plasma. *Thromb Res* 2002;107(suppl):S53-S57.

NHLBI Consensus Panel: transfusion alert: indications for the use of red blood cells, platelets, and fresh frozen plasma. Available at: www.nhlbi.nih.gov/health/prof/blood/transfusion/transfin.htm. Accessed June 14, 2007.

RATIONALE (9)

Answer: C

The clinical findings in association with the large lung mass are suggestive of hypercalcemia. The main clinical manifestations of hypercalcemia are gastrointestinal (anorexia, nausea, vomiting, abdominal pain, constipation), cardiovascular (hypertension, prolonged PR and QRS intervals, shortened QT interval, bradyarrhythmias), renal (polyuria, nephrocalcinosis), neurologic (apathy, lethargy, coma), and skeletal (bone pain).

Hypercalcemia due to lung cancer is usually associated with nonsmall cell tumors, and the most common mechanism is parathyroid hormone-related peptide. Other mechanisms of hypercalcemia associated with malignancy include ectopic production of vitamin D and bone metastasis. The initial step in management of severe hypercalcemia involves replacing intravascular volume with isotonic saline to increase renal blood flow and enhance calciuresis. After volume expansion is achieved, administration of a loop diuretic to increase renal excretion of calcium may help. Potassium and magnesium will usually require replacement during diuresis.

Additional specific therapy is usually required with extreme elevations of calcium and includes biphosphonates and calcitonin. Glucocorticoids may be helpful in hypercalcemia associated with excess vitamin D, such as granulomatous diseases or in patients with hematologic malignancy, such as lymphoma or multiple myeloma.

Although phosphate is effective in lowering the serum calcium concentration, this therapy may lead to the precipitation of calcium-phosphate complexes and is not recommended as initial therapy in hypercalcemia.

#### **REFERENCES (9)**

Body JJ, Bouillon R. Emergencies of calcium homeostasis. Rev Endocr Metab Dis 2003;4:167-175.
Grill V, Martin TJ. Hypercalcemia of malignancy. Rev Endocr Metab Dis 2000;1:253-263.
Krimsky WS, Behrens RJ, Kerkvliet GJ. Oncologic emergencies for the internist. Cleve Clin J Med 2002;69:209-217.

Nelson KA, Walsh D, Abdullah O, et al. Common complications of advanced cancer. *Semin Oncol* 2000;27:34-44.

Stewart AF. Hypercalcemia associated with cancer. N Engl J Med 2005;352:373-379.

RATIONALE (10) Answer: A

This patient's clinical history is highly suggestive of acute chest syndrome associated with sickle cell disease. Adult patients typically develop the syndrome 2 to 3 days after hospitalization for a vasoocclusive crisis. The most common clinical manifestations are fever, cough, tachypnea, chest pain, and a decrease in oxygen saturation. The etiology of the syndrome may be due to infection (most commonly chlamydia and mycoplasma), fat embolism, or atelectasis due to respiratory splinting.

Patients should receive oxygen therapy as needed and antibiotics to cover for typical pathogens (a cephalosporin and macrolide or quinolone). Exchange transfusion would be preferred in this patient over simple transfusion due to his higher hemoglobin. Simple and exchange transfusions are capable of improving oxygenation. Exchange transfusion aims to decrease hemoglobin S to <30 to 50% in patients. The hemoglobin should be maintained at  $\le 11$  g/dL to avoid hyperviscosity, which could result in additional complications such as stroke.

Exchange transfusion is often preferred in patients who have more severe diseases, multilobe involvement, persistent or worsening hypoxemia, neurologic abnormalities or multiorgan failure. Leukocyte-poor blood should be used in these patients.

Although dexamethasone (0.3 mg/kg) was shown to reduce the length of hospitalization in children with acute chest syndrome, a rebound effect resulted in increased numbers of readmission in the treatment group. No studies are available in adults, and use of dexamethasone should be considered experimental. Bronchoalveolar lavage should be reserved for those patients who fail to respond to initial therapy.

Although there are anecdotal reports of use of inhaled nitric oxide in mechanically ventilated patients, this application is also experimental and should be reserved for patients with the most severe diseases which are unresponsive to other mechanical ventilation techniques.

#### REFERENCES (10)

Ballas SK. Sickle cell anemia: progress in pathogenesis and treatment. *Drugs* 2002; 62:1143-1172. Danielson CSM. The role of red blood cell exchange transfusion in the treatment and prevention of complications of sickle cell disease. *Ther Apher* 2002;6:24-31.

Stuart MJ, Setty BNY. Acute chest syndrome of sickle cell disease: new light on an old problem. *Curr Opin Hematol* 2001;8:111-122.

Vichinsky EP, Neumayr LD, Earles AN, et al. Causes and the outcomes of the acute chest syndrome and sickle cell disease. *N Engl J Med* 2000;342:1855-1865.

RATIONALE (11) Answer: D

One of the approved indications for use of recombinant factor VIIa is serious bleeding in hemophiliacs with inhibitors. Factor VIIa enhances thrombin generation, leading to hemostasis. Factor VIII in any form (cryoprecipitate, plasma-derived or recombinant) would be ineffective due to the presence of inhibitors. Factor XI requires factor VIII as a cofactor to activate factor X. Fresh frozen plasma does not contain significant amounts of factor VII to allow sufficient thrombin generation for effective clotting. Activated prothrombin complex concentrates are another hemostatic option for hemophiliacs with inhibitor levels.

#### **REFERENCES (11)**

- Abshire T, Kenet G. Recombinant factor VIIa: review of efficacy, dosing regimens and safety in patients with congenital and acquired factor VIII or IX inhibitors. *J Thromb Haemost* 2004;2:899-909.
- Goodnough LT, Lublin DM, Zhang L, et al. Transfusion medicine service policies for recombinant factor VIIa administration. *Transfusion* 2004;44:1325-1331.
- Levi M, Peters M, Buller HM. Efficacy and safety of recombinant factor VIIa for treatment of severe bleeding: a systematic review. *Crit Care Med* 2005;33:883-890.
- Monahan PE, Aldedort LM. Factors affecting choice of hemostatic agent for the hemophilia patient with an inhibitor antibody. *Am J Hematol* 2004;77:346-350.

RATIONALE (12)

Answer: A

This patient's presentation is consistent with tumor lysis syndrome (TLS), which results from the rapid destruction of malignant cells and release of intracellular ions, nucleic acids, proteins and metabolites into the extracellular space. This process can lead to hyperuricemia, hyperkalemia, hyperphosphatemia, hypocalcemia, and uremia, leading to acute renal failure in some cases.

TLS usually occurs in patients with large tumor burdens or highly proliferative malignancies, particularly Burkitt lymphoma, high grade non-Hodgkin lymphomas, and acute leukemias. However, TLS can occur in patients usually considered to be at low risk for the syndrome, as in this patient with chronic lymphocytic leukemia. The best approach is to prevent the development of TLS, if possible, is by identifying patients at high risk and initiating hydration. It's also important to prevent hyperuricemia, which can lead to obstructive uropathy with prophylactic allopurinol treatment.

If TLS is diagnosed after initiation of therapy, vigorous IV hydration should be instituted as soon as the syndrome is recognized. Urine output should be maintained at 100 to 200 mL/h. Allopurinol is a xanthine analog that is a competitive inhibitor of xanthine oxidase, which inhibits the metabolism of purines (xanthine and hypoxanthine) to uric acid.

Immediate dialysis is not indicated, unless there is failure of medical management and/or the development of life-threatening electrolyte abnormalities despite treatment. Hemodialysis is superior to continuous renal replacement techniques for the correction of such abnormalities.

Diuretics would not be indicated initially until intravascular volume has been repleted. Urinary alkalinization (pH >7.0) rather than acidification increases the solubility of uric acid. Calcium and phosphate can precipitate at an alkaline pH and worsen renal failure so alkalinization using sodium bicarbonate should be used judiciously.

Allopurinol has several limitations in the treatment of TLS because it only prevents new uric acid formation. An alternative approach is to promote the catabolism of uric acid to allantoin by the use of rasburicase, a recombinant form of urate oxidase. Rasburicase has been used for the prevention and treatment of hyperuricemia associated with TLS. This medication should be avoided in patients with glucose-6-phosphate dehydrogenase deficiency.

#### **REFERENCES (12)**

Cairo MS, Bishop M. Tumor lysis syndrome: new therapeutic strategies and classifications. Br J Haem 2004;127:3-11.

Davidson MB, Thakkar S, Hix JK, et al. Pathophysiology, clinical consequences, and treatment of tumor lysis syndrome. *Am J Med* 2004;116:546-554.

RATIONALE (13)

Answer: E

This patient most likely has a transfusion-related acute lung injury (TRALI). TRALI is defined as an acute lung injury (P/F <300) temporally related to the transfusion of blood products, and it has been reported to be the leading cause of transfusion-related death in the United States. Additional clinical criteria for diagnosis include bilateral fluffy infiltrates consistent with pulmonary edema, pulmonary artery occlusion pressure ≤18 mm Hg, and no clinical evidence of left atrial hypertension. TRALI usually develops within 6 hours of exposure to blood products. Clinical findings include tachypnea, dyspnea, cyanosis, fever, and acute hypoxemia. Treatment includes aggressive respiratory support with supplemental oxygen, and mechanical ventilation if needed. With supportive care, most patients recover within 72 hours. There is no role for corticosteroids or diuretics.

All plasma-containing blood products can be associated with TRALI, but platelets are the most commonly implicated, followed by fresh frozen plasma and red blood cells. Washed red cells would eliminate the potential risk for TRALI.

Several mechanisms have been proposed for TRALI. The single-event hypothesis is the passive transfusion of donor antibodies directed against recipient antigens on their leukocytes, or the transfusion of donor leukocytes with recipient antibodies directed against antigens on the donor cells. The two-event hypothesis suggests that the underlying patient condition leads to pulmonary sequestration of neutrophils; transfusion is the second event, involving donor antibodies to the neutrophils or other biologic response modifiers.

Pulmonary embolism, although possible, is not consistent with the radiographic findings. No information is given to suggest infection. Bacterial contamination of blood products can occur but is rare and usually manifests as fever, hypotension and circulatory collapse. Transfusion associated circulatory overload is a common complication of transfusion but the hemodynamic data provided do not suggest volume overload.

#### **REFERENCES (13)**

- Gajic O, Moore SB. Transfusion-related acute lung injury. *Mayo Clin Proc* 2005;80:766-770. Looney MR, Gropper MA, Matthay MA. Transfusion-related acute lung injury. A review. *Chest* 2004;126:249-258.
- Silliman CC, Ambruso DR, Boshkov LK. Transfusion-related acute lung injury. *Blood* 2005;105:2266-2273.
- Toy P, Popovsky MA, Abraham E, et al. Transfusion-related acute lung injury: definition and review. Crit Care Med 2005;33:721-726.

RATIONALE (14)

Answer: B

The anemia, large protein gap and hepatosplenomegaly suggest the presence of a plasma cell dyscrasia such as Waldenstrom macroglobulinemia. Pending results of serum and urine protein electrophoresis, the deterioration in the patient's neurologic status is likely a manifestation of a hyperviscosity syndrome. Hyperviscosity syndrome can occur with myelomas, chronic myelogenous leukemia (CML), polycythemia vera, cryoglobulinemias, rheumatoid arthritis, and, rarely, with other connective tissue disorders. Waldenstrom's macroglobulinemia is more often associated with this syndrome than with other myelomas, due to the large size of the monoclonal immunoglobulin M molecules, which have a high intrinsic viscosity. High levels of immunoglobulin G and immunoglobulin A can result in the formation of aggregates that also increase viscosity.

Hyperviscosity in CML is due to the high number of white blood cells (often >300,000/mm³), and to the increased number of red blood cells in polycythemia. In patients with myelomas who have manifestations of significant bleeding or ischemia (central nervous system or cardiovascular), the treatment of choice is plasmapheresis to remove paraproteins. Plasmapheresis may be required 2 to 3 times to control viscosity as disease-specific treatment is initiated.

Thrombolytics would not be appropriate in a patient with possible hyperviscosity syndrome, since the neurologic symptoms are not due to thrombosis. In addition, patients with hyperviscosity have an increased incidence of mucosal bleeding. Transfusion should not be performed initially, as it will further increase viscosity. Corticosteroids may impact the myeloma but would not relieve the symptoms of hyperviscosity.

#### **REFERENCES (14)**

- Della Rossa A, Tavoni A, Bombardieri S. Hyperviscosity syndrome in cryoglobulinemia: clinical aspects and therapeutic considerations. *Semin Thromb Hemost* 2003;29:473-477.
- Mehta J, Singhal S. Hyperviscosity syndromes in plasma cell dyscrasias. *Semin Thromb Hemost* 2003; 29:467-471.
- Rampling MW. Hyperviscosity as a complication in a variety of disorders. *Semin Thromb Hemost* 2003;29:459-465.

RATIONALE (15)

Answer: B

Because the progression of infection in neutropenic patients can be rapid and mortality is high when untreated, empirical antibiotic therapy should be initiated promptly to all neutropenic patients at the onset of fever. When choosing empirical antimicrobial regimens, it is crucial to understand the institution-predominant pathogens and resistance patterns, as well as individual patient characteristics. Currently, monotherapy with the newer broad-spectrum antimicrobials has become the norm. Empirical administration of glycopeptides, such as vancomycin, without documentation of a grand positive infection, is currently not favored at the onset of empirical therapy. Combination therapy with a double gram negative coverage, although utilized in the past, has lost favor secondary to the appearance of new broad-spectrum antibiotics and some reports of worse outcomes with combination regimens, most likely secondary to complications from aminoglucacytes.

The addition of empirical antifungal coverage is not recommended at the beginning of therapy. However, if fever does not resolve and the patient remains neutropenic after a period of 3-5 days, some authors recommend the introduction of both glycopeptides and/or antifungal agents such as fluconazole.

Of the given options in this question, the correct answer is ceftazidime. There in no indication for double-gram negative coverage, although the patient has a porta catheter, as there is no evidence of catheter-related infection at this point. Finally, it is also very important to remember that if positive cultures or sites of infections are identified, appropritate antibiotics modifications should be made.

#### **REFERENCES (15)**

Sipsas NV, Bodey GP, Kontoyiannis DP. Perspectives for the management of febrile neutropenic patients with cancer in the 21st century. *Cancer* 2005;103:1103-1113.

Hughes WT, Armstrong D, Bodey GP, et al. 2002 guidelines for the use of antimicrobial agents in neutropenic patients with cancer. Clin Infect Dis 2002;34:730-751.

## SECTION 6: Hemodynamic Monitoring

## SECTION 6: HEMODYNAMIC MONITORING

Instructions: For each question, select the most correct answer.

1. A 40-year-old female with a history of metastatic carcinoma of the breast is admitted to the ICU with hypotension (BP 65/40 mm Hg, HR 110/min, RR 38/min). She received several fluid boluses before a pulmonary artery catheter is inserted. The following hemodynamic values were obtained: pulmonary artery pressure 40/28 mm Hg, central venous pressure 26 mm Hg, pulmonary artery occlusion pressure 26 mm Hg, cardiac index 1.9, stroke volume 25 mL.

Which one of the following is the most likely explanation for the low cardiac index?

- A. Pneumothorax
- B. Hypovolemia
- C. Cardiac tamponade
- D. Congestive heart failure
- E. Malignant pleural effusion
- 2. Which one of the following is most useful for predicting accurate arterial line assessment of systemic blood pressure prior to the insertion of an arterial catheter in the radial artery?
  - A. Modified Allen's test
  - B. Noninvasive blood pressure measurements from both arms
  - C. Allen's test
  - D. Doppler assessment
  - E. Ultrasound

3. A 76-year-old woman, 4'10" tall, is admitted to the ICU with hypotension and suspected sepsis. She is intubated. Her past medical history is pertinent for a remote above-the-knee amputation for trauma. Her systemic BP is 100/58 mm Hg, supported with an infusion of norepinephrine at 10 μg/min, and her HR is 98. A pulmonary artery catheter is inserted. An occlusion pressure is obtained with balloon inflation from an insertion distance of 39 cm. The following results were obtained: pulmonary artery 32/18 mm Hg, central venous pressure 10 mm Hg, pulmonary artery occlusion pressure 10 mm Hg, cardiac index 7.

Which of the following is most likely?

- A. The cardiac index of 7 is secondary to the B, effects of the norepinephrine
- B. The cardiac index of 7 is consistent with a hyperdynamic physiology secondary to sepsis
- C. The cardiac output determined by the thermodilution method may be overestimated for this patient
- D. The cardiac index of this patient is based on accurate data
- E. In this patient, there is no decrease of cardiac output from decreased venous return associated with mechanical ventilation
- 4. A 43-year-old male is admitted to the ICU following extraction from an airplane fire. He was intubated in the field for presumed smoke inhalation. Arterial blood gas was 7.26/44/210, while supported with Fio, 0.65, pressure control 20, rate 12, and positive end expiratory pressure 8 cm H<sub>2</sub>O.

Which one of the following is the best determinant of the presence of carbon monoxide poisoning?

- A. Pulse oximetry
- B. Mixed venous oxygen saturation
- C. Co-oximetry
- D. Capnography
- E. Neurologic examination

5. A 70-year-old male is admitted to the ICU following an aortic valve replacement for aortic stenosis. A pulmonary artery catheter was placed preoperatively. His postoperative hemodynamic data are as follows: BP 132/74 mm Hg, HR 70/min, pulmonary artery pressure 32/16 mm Hg, central venous pressure 8 mm Hg, and pulmonary artery occlusion pressure 8 mm Hg. Two hours after admission, you are called to the bedside, because he has had frequent episodes of ventricular tachycardia with a stable blood pressure.

Which of the following is the most appropriate next step?

- A. Amiodarone, 150 mg IV
- B. Lidocaine, 100 mg IV
- C. Confirm the position of the pulmonary artery catheter
- D. Quickly remove the pulmonary artery catheter
- E. Observe the patient without intervention
- 6. A 32-year-old mesomorphic female competitive tennis player has a history of a total colectomy for ulcerative colitis and is now pregnant at 36 weeks gestation. She is admitted to the ICU following the lysis of adhesions for a small bowel obstruction. Initial empiric fluid administration was several liters of crystalloid, with the assumption that her tachycardia was secondary to a relative hypovolemia. When the tachycardia did not resolve, a pulmonary artery catheter was placed, with the following values resulting: central venous pressure 5 mm Hg, pulmonary artery occlusion pressure 5 mm Hg, cardiac output 16 LPM, systemic vascular resistance 250 dyne·sec/cm<sup>5</sup>.

Which one of the following is most correct?

- A. The systemic vascular resistance indicates that the patient has distributive shock
- B. The patient is hypertensive
- C. The elevated cardiac output is overestimated based on error
- D. The degree of cardiac output elevation is consistent with pregnancy
- 7. Using the modified Seldinger technique for placement of a central venous catheter, what is the most accurate way to determine if a hollow bore needle is placed in the jugular vein and not the carotid artery before the insertion of a guide wire?
  - A. Color of the blood withdrawn from the vessel
  - B. The absence of pulsatile flow in the vessel
  - C. Palpation of the carotid artery during insertion of the needle into the vein
  - D. Initial placement of the needle with ultrasound guidance
  - E. Transduction of the vascular pressure from the needle prior to insertion of the guide wire

8. A 60-year-old male with chronic renal insufficiency and hypertension is admitted to the ICU after an uneventful abdominal aortic aneurysm repair. His initial hemodynamic data are as follows: BP 140/60 mm Hg, HR 50/min, RR 18/min, cardiac output (CO) 3.2 LPM, pulmonary arterial occlusion pressure (PAOP) 12 mm Hg.

Because of a low urine output, a 500 mL bolus of a colloid is given. After the fluid bolus, which one of the following best indicates that this patient has diastolic dysfunction?

9	CO, LPM	PAOP, mm Hg
A.	3.4	14
B.	4.2	14
C.	3.8	18
D.	5.0	12
E.	4.2	16

- 9. A 60-year-old woman with a history of hypertension, severe aortoiliac disease (status post aortobifemoral bypass graft), atrial fibrillation, and previous replacement of her mitral valve is admitted to the ICU following an Ivor-Lewis esophagectomy (combined laparotomy and right thoracotomy). She was extubated that evening, and a heparin infusion was started in lieu of her usual warfarin anticoagulation. Although she received several boluses of fluid and there was no evidence of bleeding, she continued to have a modest pressor requirement. At this time, what would be the best method to determine her cardiac performance?
  - A. Pulmonary artery catheter
  - B. Transesophageal echocardiogram
  - C. Cardiac output via esophageal Doppler
  - D. Indirect Fick method using CO, rebreathing

10. A 62-year-old male is being treated with a nitroglycerin infusion and heparin anticoagulation after an acute myocardial infarction. After several failed attempts to insert the arterial catheter in both radial arteries, an alternate arterial site is considered.

Which one of the following is correct?

- A. Cannulation of the axillary artery in this patient would be preferred over the brachial artery because the axillary artery has a better collateral circulation
- B. The brachial artery has a greater margin of safety compared to the axillary artery in an anticoagulated patient
- C. After a failed radial artery attempt, cannulation of the ulnar artery in the same wrist would be safe as long as the radial artery was not punctured
- D. A well-known complication of brachial artery cannulation is damage to the brachial plexus
- 11. A 57-year-old female with sepsis is admitted to the ICU on postoperative day 3, following a right pneumonectomy. The critical care team has decided to place a pulmonary artery catheter (PAC) to help with fluid management and pressor administration. The PAC will easily float into the stump of the right pulmonary artery. Which of the following is correct?
  - A. All of the cardiac output is going to this side
  - B. The safest method of placement of the PA catheter in this patient is with fluoroscopic guidance or echocardiography
  - C. An insertion of the PAC into the femoral vein is preferred in pneumonectomy patients
  - D. The risk of subclavian artery puncture is lessened on the pneumonectomy side of the patient
- 12. When the height of a patient's bed is changed, what is appropriate to assure accuracy of an arterial and pulmonary artery catheter transducers?
  - A. Confirm that the transducers are zeroed at the new height
  - B. Confirm that the transducers are at the level of the right atrium
  - C. Perform a "pop" test at the new bed height
  - D. Check the pulmonary artery occlusion pressure at the new height

13-16. Use the table below to answer questions 13-16.

. Match the correct hemodynamic profiles below with the clinical information. Letters may be used more than once.

	Cardiac Index (L/m²/min)	Stroke Volume (mL)	Central Venous Pressure	Pulmonary Artery Occlusion Pressure (mm Hg)
28	,		(mm Hg)	
A.	1.5	20	2	2
B.	1.5	25	16	18
C.	1.9	40	19	8
D.	7.2	102	6	6

- 13. Biventricular failure
- 14. Hypovolemia
- 15. Hepatic failure
- 16. Right heart failure
- 17. Following a right colectomy, a patient who is a Jehovah's witness is admitted to the ICU. She remains intubated and supported with mechanical ventilation. The plan is to minimize laboratory tests to reduce the blood draws. Pulse oximetry and capnography are used to assess oxygenation and ventilation. Her oxyhemoglobin saturation is 90%. Which one of the following most closely approximates the PaO<sub>2</sub>?
  - A. 60 mm Hg
  - B. 75 mm Hg
  - C. 90 mm Hg
  - D. 100 mm Hg
  - E. This cannot be made unless the F10, is provided

- 18. A 75-year-old woman arrives in the ICU at 0930 hrs for postoperative care after a total abdominal hysterectomy. She is 60 inches tall and weights 50 kg. Noninvasive BP is being measured. The BP when she left the operating room was 130/80, compared with 96/62 that was initially obtained in the ICU. There has been no change in BP over the next 30 minutes. Her other vital signs are stable: HR is 68, RR 16, Spo<sub>2</sub> 98% on 2 L/min oxygen via nasal cannula. Intraoperatively, the estimated blood loss was 300 mL, she was given 2,000 mL lactated Ringer, the urine output for the 2-hour procedure was 150 mL, and the intraoperative hemoglobin at the end of the procedure was 8.2 mg/dL. What is the most appropriate intervention?
  - A. Transfusion of 1 U packed red blood cells
  - B. Placement of an arterial catheter
  - C. Confirmation that an appropriate-sized blood pressure cuff is being used
  - D. Further observance for decreases in blood pressure
  - E. Increase the blood pressure using a phenylephrine infusion
- 19. A 45-year-old male involved in a motor vehicle collision has a closed head injury. His blood pressure is monitored with an arterial line. Intracerebral pressure is being monitored with a goal of a cerebral perfusion pressure of 70-80 mm Hg. The head of the bed is elevated 30°. Which one of the following is correct?
  - A. The arterial transducer must be level to the Circle of Willis
  - B. The arterial transducer must be level to the right atrium
  - C. Jugular venous pressure monitoring is a requirement
  - D. If the transducer is moved from the level of the right atrium to the Circle of Willis, it must be rezeroed for accuracy
  - E. The use of an intracerberal pressure monitor lessens the need for precise arterial pressure monitoring

#### 20-24. For questions 20-24, please use the following clinical scenario:

A 66-year-old woman is admitted to the ICU with suspected biliary sepsis. She was profoundly hypotensive on arrival to the hospital, and, after aggressive crystalloid resuscitation, she has persistently low arterial blood pressure (78/40 mm Hg). As crystalloid resuscitation continues, a pulmonary artery catheter is inserted. The following data are obtained:

Arterial blood pressure, mm Hg	88/42
Mean arterial pressure, mm Hg	57
Heart rate, beats/min	108
Central venous pressure, mm Hg	10
Pulmonary capillary wedge pressure, mm Hg	18
Cardiac index, L/min/m <sup>2</sup>	2.1
Systemic vascular resistance, dynes/sec/cm <sup>5</sup>	895

A vasoactive drug is administered. In the next 5 questions, sets of hemodynamic parameters will be provided. Match each set of hemodynamic parameters in each question with the best choice for vasoactive drug administered.

- A. Dopamine at 8 μg/kg/min
- B. Dopamine at 25 μg/kg/min
- C. Dobutamine
- D. Norepinephrine
- E. Phenylephrine

20. Arterial blood pressure, mm Hg	99/48
Mean arterial pressure, mm Hg	65
Heart rate, beats/min	120
Central venous pressure, mm Hg	8
Pulmonary capillary wedge pressure, mm Hg	15
Cardiac index, L/min/m <sup>2</sup>	3.0
Systemic vascular resistance, dynes/sec/cm <sup>5</sup>	760

### Self-Assessment in Multiprofessional Critical Care

21 Arterial blood programs II-	120/65
21. Arterial blood pressure, mm Hg Mean arterial pressure, mm Hg	120/65
Heart rate, beats/min	83 125
Central venous pressure, mm Hg	8
Pulmonary capillary wedge pressure, mm Hg	15
Cardiac index, L/min/m <sup>2</sup>	3.1
Systemic vascular resistance, dynes/sec/cm <sup>5</sup>	968
•	
	<del></del>
22. Arterial blood pressure, mm Hg	120/65
Mean arterial pressure, mm Hg	83
Heart rate, beats/min	80
Central venous pressure, mm Hg	12
Pulmonary capillary wedge pressure, mm Hg	20
Cardiac index, L/min/m <sup>2</sup>	1.8
Systemic vascular resistance, dynes/sec/cm <sup>5</sup>	1,578
23. Arterial blood pressure, mm Hg	130/70
Mean arterial pressure, mm Hg	90
Heart rate, beats/min	100
Central venous pressure, mm Hg	10
Pulmonary capillary wedge pressure, mm Hg	16
Cardiac index, L/min/m <sup>2</sup>	2.5
Systemic vascular resistance, dynes/sec/cm <sup>5</sup>	1,280
	· <del>/</del>

24. Arterial blood pressure, mm Hg Mean arterial pressure, mm Hg Heart rate, beats/min Central venous pressure, mm Hg Pulmonary capillary wedge pressure, mm Hg Cardiac index, L/min/m²	80/36 51 120 8 15 3.3
Cardiac index, L/min/m <sup>2</sup>	3.3
Systemic vascular resistance, dynes/sec/cm <sup>5</sup>	521

25. A 72-year-old female patient is admitted with peritonitis. She has an exploratory laparotomy which identifies a bowel perforation that is repaired. In the postanesthesia care unit, the patient remains on a ventilator and develops hypotension. Over the course of several hours, she manifests septic shock requiring high dose vasopressors. She has evidence of hypoperfusion with cool extremities and lactic acidosis. An arterial blood gas analysis shows a pH 7.24, Pco<sub>2</sub> 28 mm Hg, PaO<sub>2</sub> 81 mm Hg, arterial saturation 97%. The hemoglobin concentration is 8.0 g/dL. A pulmonary artery catheter is inserted, and the following measurements are obtained: central venous pressure 10 mm Hg, pulmonary capillary wedge pressure 18 mm Hg, cardiac index 1.5 L/min/m², and mixed venous oxygen saturation of 54%.

Which of the following interventions is most likely to maximally increase oxygen delivery?

- A. 2 L of crystalloid infusion through large bore IV lines
- B. Transfuse the patient to a hemoglobin of 10 g/dL
- C. Administer an inotrope to achieve a cardiac index of 3.0 L/min
- D. Increase the fraction of inspired oxygen concentration and/or positive end expiratory pressure to achieve a higher PaO,

26. A patient with a history of chronic obstructive pulmonary disease and obstructive sleep apnea is brought to the ICU from the medical ward because of shock of unclear etiology. You continue aggressive crystalloid resuscitation as you insert a pulmonary artery catheter. The central venous pressure is noted to be in a range that you believe indicates hypovolemia for this patient. The patient is still hypotensive. You ask the nurse to continue crystalloid fluid boluses because you want to optimize cardiac preload. Referring to the pulmonary capillary wedge pressure (PCWP), the ICU nurse asks you: "Where would you like the wedge to be located?"

Which of the following statements is most correct?

- A. A PCWP of 10-12 mm Hg should be targeted
- B. A PCWP of 18-20 mm Hg should be targeted
- C. The optimal pulmonary capillary wedge pressure to maximize preload is unknown
- D. Crystalloid boluses should be given only until the blood pressure fails to rise with continued administration
- 27. Which one of the following suggests that the pulmonary artery catheter is incorrectly positioned for measurement of pulmonary artery occlusion pressure (PAOP)?
  - A. Catheter tip is below left atrium on chest radiograph
  - B. Blood withdrawn from the tip of the catheter with the balloon up has 97% O<sub>2</sub> saturation and the arterial O<sub>3</sub> saturation is 94%
  - C. Pulmonary artery pressure 24/12 mm Hg and PAOP 14 mm Hg
  - D. Blood is withdrawn freely from the catheter

28. A patient in the ICU is currently on mechanical ventilation with sedation and neuromuscular blockade. The patient has the waveforms shown in Figure 1 below.

Which of the following statements is most correct regarding this patient?

- A. This patient has cardiac tamponade
- B. Administration of a fluid bolus is likely to increase the cardiac output
- C. Administration of a fluid bolus is likely to cause pulmonary edema
- D. This patient has high airway resistance

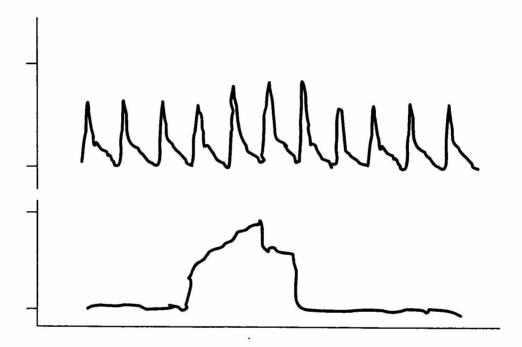
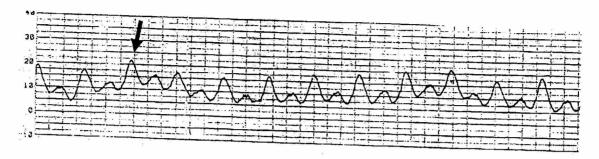
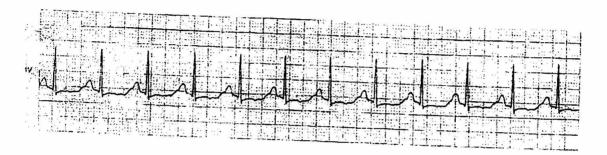


Figure 1. PA = arterial pressure, PAW = airway pressure

29. A 45-year-old female is mechanically ventilated 12 hours following cardiovascular surgery. She is heavily sedated and not triggering the ventilator.

Which one of the following statements concerning the pulmonary artery occlusion pressure (PAOP), as shown in the figure below, is most correct?





- A. The arrow points to the v-wave and PAOP is approximately 8 mm Hg
- B. The arrow points to the a-wave and PAOP is approximately 8 mm Hg
- C. The arrow points to the v-wave and PAOP is approximately 14 mm Hg
- D. The arrow points to the a-wave and PAOP is approximately 14 mm Hg

## **SECTION 6: HEMODYNAMIC MONITORING**

#### **ANSWERS:**

1-C; 2-B; 3-C; 4-C; 5-C; 6-B; 7-E; 8-C; 9-A; 10-B; 11-A; 12-B; 13-B; 14-A; 15-D; 16-C; 17-A; 18-C; 19-A; 20-A; 21-B; 22-E; 23-D; 24-C; 25-C; 26-C; 27-C; 28-B; 29-A

RATIONALE (1)

Answer: C

Carcinoma of the breast is one of the most common causes of pericardial tamponade. Other causes include bronchogenic carcinoma, lymphoma, renal failure, and tuberculosis. The physiologic manifestations of a tamponade are the elevation of the right atrial mean, the right ventricular diastolic, the pulmonary artery diastolic, and the pulmonary artery occlusion pressures, with values within 2 to 3 mm Hg of each other.

If not recognized, cardiac tamponade can rapidly become fatal, and should be considered for any patient with shock or pulseless electrical activity. Medical management is usually ineffective; fluid administration may be of only temporary benefit to the hypovolemic patient while definitive treatment is arranged.

Echocardiographic pericardiocentesis can be performed at the bedside. Definitive treatment for persistent cardiac effusions is a surgical pericardiectomy through a sub-xyphoid incision.

#### **REFERENCES (1)**

Collins D. Aetiology and management of acute cardiac tamponade. Crit Care Resusc 2004;6:54-58. Little WC, Freeman GL. Pericardial disease. Circ J Am Heart Assoc 2006;113:1622-1632.

Molina Garrido MJ, Mora Rufete A, Rodrequez-Lescure A, et al. Recurrent pericardial effusion as initial manifestation of primary diffuse pericardial malignant mesothelioma. *Clin Transl Oncol* 2006;8:694-696.

Nagarsheth NP, Harrison M, Kalir T, Rahaman J. Malignant pericardial effusion with cardiac tamponade in a patient with metastatic vaginal adenocarcinoma. *Int J Gynecol Cancer* 2006;16:1458-1461.

RATIONALE (2)

Answer: B

The original and modified Allen's tests, Doppler, and ultrasound will provide data on arterial flow and collateral circulation but not pressure. There are many conditions, such as thoracic aortic dissection and coarctation of the aorta, that may lead to differences in the blood pressure measurements between the upper extremities. In addition, in normal individuals with no known vascular pathology, it has been reported that about half have systolic or diastolic interarm differences of >10 mm Hg, and almost 20% of these had differences >20 mm Hg. For this reason, noninvasive blood pressure measurements should be obtained from both arms. If there is a significant difference in pressures, the arterial catheter is ideally placed in the extremity with the higher pressure.

#### **REFERENCES (2)**

- Barbeau GR, Arsenault F, Dugas L, Simard S, Lariviere MM. Evaluation of the ulnopalmar arterial arches with pulse oximetry and plethysmography: comparison with the Allen's test in 1010 patients. *Am Heart J* 2004;147:489-493.
- Jarvis MA, Jarvis CL, Jones PR, Spyt TJ. Reliability of Allen's test in selection of patients for radial artery harvest. *Ann Thorac Surg* 2000;70:1362-1365.
- Oettle AC, van Niekerk A, Boon JM, et al. Evaluation of Allen's test in both arms and arteries of left and right-handed people. Surg Radiol Anat 2006;28:3-6.
- Schlichtig RI. Arterial catheterization: complications. In Tobin MJ, ed. *Principles and Practice of Intensive Care Monitoring*. New York, NY: McGraw-Hill; 1998, 751-756.
- Singer AJ, Hollander JE. Blood pressure: assessment of inter-arm differences. *Arch Intern Med* 1996;156:2005-2008.

RATIONALE (3)

Answer: C

At a wedge position of 39 cm, the central venous pressure port through which the cardiac output injectate flows is in proximity to the side arm of the introducer. Some of the injectate will reflux into the side arm and will not be read by the thermister at the distal end of the pulmonary artery catheter. A critical value needed in the routine set up for cardiac output measurements is the size of the injectate, normally 10 mL for adults. This is used in the following calculation: Cardiac output = quantity of indicator/(c)(t) where c is the average concentration of the injectate during the first pass, and it is the total duration of the interval of injectate that is sensed.

When the cardiac output is low, the time that the injectate is sensed will be prolonged. When the cardiac output is elevated, this interval is shortened. Therefore, a smaller injectate will have a shorter transit time, and the cardiac output will be calculated at a higher value than it actually is. The cardiac index, which is cardiac output/body surface area, corrects the cardiac output for differences in body size.

The original formula for body surface area was derived in 1916 from direct measurements made from molds of only nine individuals of varying age, shape, and size. A more simple formula was developed recently: body surface area  $(m^2) = [height (cm) \times (kg)]/3,600)^{1/2}$ . Therefore, it is a derived value based on several assumptions. In this patient, it will underestimate cardiac performance, since it will not compensate for the loss of surface area of the lower extremity.

#### **REFERENCES (3)**

DuBois D; DuBois EF. A formula to estimate the approximate surface area if height and weight be known. *Arch Int Med* 1916;17:863-871

Mosteller RD. Simplified calculation of body surface area. *N Engl J Med* 1987:22;317:1098 Stoller JK, et al. Spurious high cardiac output from injecting thermal indicator through an ensheathed port. *Crit Care Med* 1986;14:1064-1065.

RATIONALE (4)

Answer: C

In this setting, both carbon monoxide (from smoke inhalation) and cyanide (from inhalation of cyanide-containing combustion products) toxicities are likely. Oxyhemoglobin saturations would be falsely elevated since the absorption spectrum of carboxyhemoglobin and oxyhemoglobin are similar. These can be distinguished with co-oximetry arterial blood gas analysis. Mixed venous oxygenation may be elevated in both through the blockade of cytochrome oxidase. Neurologic examination, coupled to a high index of suspicion, is one of the best methods to diagnosis carbon monoxide poisoning, but would not be useful in this intubated patient. There is no role for capnography.

#### **REFERENCES (4)**

Hampson NB. Pulse oximetry in severe carbon monoxide poisoning. *Chest* 1998;114:1036-1041. Hampson NM, Scott KL, Zmaeff JL. Carboxyhemoglobin measurement by hospitals: implications for the diagnosis of carbon monoxide poisoning. *J Emerg Med* 2006;31:13-16.

Mokhlesi B, Leikin JB, Murray P, Corbridge TC. Adult toxicology in critical care. Part II. Specific poisonings. *Chest* 2003;123:897-922.

Varon J, Marik PE, Fromm RE, Gueler A. Carbon monoxide poisoning: a review for clinicians. J Emerg Med 1999;17:87-93.

Vender JS, Szokol JW. Hemodynamic assessment in the critically ill patient. In Murray MJ, Coursin DB, Pearl RG, Prough DS, eds. *Critical Care Medicine* Philadelphia, PA: Lippincott; 2002, 122-136.

RATIONALE (5)

Answer: C

This patient has stable ventricular tachycardia. Ventricular dysrhythmia are often manifested when the pulmonary artery (PA) outflow tract is irritated by the tip of the PA catheter during placement. Prior to instituting drug therapy or removing the PA catheter, it is prudent to determine that the PA catheter has not been inadvertently withdrawn so that the tip is in an area of ventricular irritability.

#### **REFERENCES (5)**

Keusch DJ, Winters S, Thys DM. The patient's position influences the incidence of dysrhythmias during pulmonary artery catheterization. *Anesthesiology* 1989;70:582-584.

Preas HL and Suffredini AF. Pulmonary artery catheterization: insertion and quality control. In: Tobin MJ, ed. *Principles and Practice of Intensive Care Monitoring*. New York, NY: McGraw-Hill; 1998, 773-795.

Pulmonary artery catheter education project. Available at www.pacep.org.

Sprung CL, Jacobs LJ, Caralis PV, Karpf M. Ventricular arrythmias during Swan-Ganz catheterization of the critically ill. *Chest* 1981;79:413-415.

Sprung CL, Pozen RG, Rozanski JJ. Advanced ventricular arrythmias during bedside pulmonary artery catheterization. Am J Med 1982;72:413-415.

RATIONALE (6)

Answer: B

The systemic vascular resistance (SVR) is a derived value calculated as the following: (mean arterial pressure – central venous pressure)/cardiac output x 80 dyne sec/cm<sup>5</sup>

The normal SVR is 900-1,400 dyne-sec/cm<sup>5</sup>. However, this patient is an athlete, and responded to physiologic stresses with a much-elevated cardiac output. From the data provided, the mean areterial blood pressure is calculated as 60 mm Hg. In a near-term pregnant patient, diastolic BP is decreased to 25% of prepregnancy values, based, upon a decrease in vascular tone. Therefore, a mean arterial blood pressure of 60 mm Hg with an SVR of 250 dynes-sec/cm<sup>5</sup> does not always equate with hypoperfusion.

#### **REFERENCES (6)**

Pinsky MR. Hemodynamic profile interpretation. In Tobin MJ, ed. *Principles and Practice of Intensive Care Monitoring*. New York, NY: McGraw-Hill; 1998, 871-888.

Varon AJ. Hemodynamic monitoring: arterial and pulmonary artery catheters. In: Civetta JM, Taylor RW, Kirby RR, eds. *Critical Care*. 2nd ed. Philadelphia, PA: Lippincott; 1992, 255-270.

Yeomans ER, Gilstrap LC. Physiologic changes in pregnancy and their impact on critical care. Crit Care Med 2005;33:S256-S258.

RATIONALE (7)

Answer: E

Arterial puncture is a known complication of central venous catheterization. A color change between simultaneously drawn arterial and venous samples prior to placement of the central line may be accurate; however, it is not the most accurate.

Absence of pulsatile flow can be secondary to obstruction. Although ultrasound guidance is superior to palpation alone, it is still not confirmatory and totally reliable to exclude intra-arterial cannulation.

Placement of a small diameter catheter into the vein and transduction of the vascular pressure is the most accurate physiologic method for confirming placement.

#### **REFERENCES (7)**

- Augoustides JG, Horak J, Ochrock AE, et al. A randomized controlled clinical trial of real-time needle-guided ultrasound for internal jugular venous cannulation in a large university anesthesia department. *J Cardiothorac Vasc Anesth* 2005;19:310-315.
- DePietro M, Espositio C, Eichacker PQ. Complications of pulmonary artery catheterization. In Tobin MJ, ed. *Principles and Practice of Intensive Care Monitoring*. New York: McGraw-Hill; 1998, 855-870.
- Farrell J, Walshe J, Gellens M, Martin KJ. Complications associated with insertion of jugular venous catheters for hemodialysis: the value of postprocedural radiograph. *Am J Kidney Dis* 1997;30:690-692.
- Schummer W, Schummer C, Rose N, et al. Mechanical complications and malpositions of central venous cannulations by experienced operators: A prosepective study of 1794 catheterizations in critically ill patients. *Intensive Care Med* 2007;33:1055-1059.

RATIONALE (8)

Answer: C

Long-standing hyptertension and valvular disease may cause a nonlinear decreased left ventricular compliance through hypertrophy of the myocardium. Reduced compliance may also occur with myocardial ischemia, restrictive cardiomyopathy, tamponade, and myocardial fibrosis. After a fluid challenge, the smallest increase in cardiac output with the highest increase in the PAOP, plus the history, support the diagnosis of diastolic dysfunction.

#### **REFERENCES (8)**

- Davidson CJ, Bonow RO. Cardiac catheterization. In: Braunwald E, Zipes DP, Libby P., eds. *Heart Disease*. Vol. 1. Philadelphia, PA: WB Saunders; 2005, 359-386
- Leatherman JW, Marini JJ. Pulmonary artery catheterization: interpretation of pressure recordings. In: *Principles and practice of intensive care monitoring*. Tobin MJ, ed. New York, NY:McGraw-Hill, 1998, 821-837.
- Pinsky MR. Hemodynamic profile interpretation. In Tobin MJ, ed. *Principles and Practice of Intensive Care Monitoring*. New York, NY: McGraw-Hill; 1998, 871-888.

RATIONALE (9)

Answer: A

Cancers of the mid-esophagus can be surgically excised during an Ivor-Lewis esophagectomy through an abdominal incision and a right thoracotoomy. Following this resection, the anastomosis of the esophagus is poorly vascularized and tenuous. Placement of devices such as a transesophageal echocardiograph or esophageal Doppler probes are contraindicated. The indirect Fick method would not be reliable since the patient is not intubated.

#### **REFERENCES (9)**

- Chaney JC, Derdak S. Minimally invasive hemodynamic monitoring for the intensivist: current and emerging technology. *Crit Care Med* 2002;30:2338-2345.
- Magder S. Cardiac output. In Tobin MJ, ed. Principles and Practice of Intensive Care Monitoring. New York, NY: McGraw-Hill; 1998, 797-810.
- Singer M, Clarke J, Bennett ED. Continuous hemodynamic monitoring by esophageal Doppler. Crit Care Med 1989;17:335-336.

RATIONALE (10)

Answer: B

The brachial artery is often not considered as a site for arterial cannulation because it has a poor collateral circulation and is in proximity to the median nerve. In this patient, however, it is the best option. Excessive bleeding from a punctured axillary artery in an anticoagulated patient has the potential for damage to the brachial plexus. Failure of blood return during a catheter insertion attempt does not preclude trauma to the artery that may not be noticed because of adequate collateral circulation. Should this occur, a catheter placed into the ipsilateral ulnar artery may cause severe ischemia to the hand.

#### **REFERENCES (10)**

Barnes R, Foster E, Janssen A, et al. Safety of brachial artery catheters as monitors in the intensive care unit: prospective evaluation with the Doppler ultrasonic velocity detector. *Anesthesiology* 1976;44:260-264.

Lodato RF. Arterial pressure monitoring. In Tobin MJ, ed. Principles and Practice of Intensive Care Monitoring. New York, NY: McGraw-Hill; 1998, 733-749.

RATIONALE (11)

Answer: A

Placement of a PAC in a patient after a pneumonectomy can be technically challenging. Although the PAC is flow-directed, passage into a fresh pulmonary artery stump on the pneumonectomy side is possible with the risk of pulmonary artery rupture. Ultrasound- guided insertion of central venous catheters minimizes the risks of pneumothorax as well as inadvertent arterial puncture. The risk of subclavian artery puncture is independent upon the presence of lung tissue. There is no risk of pneumothorax with a femoral vein insertion but it is often difficult to float into the pulmonary artery using this approach. The heart and great vessels can easily be visualized with fluoroscopy and echocardiography to guide the passage of the PAC, regardless of the insertion site, into the pulmonary artery.

#### **REFERENCES (11)**

- Mallory DL, McGee WT, Shawker TH, Brenner M, et al. Ultrasound guidance improves the success rate of internal jugular vein cannulation. A prospective, randomized trial. *Chest* 1990;98:157-160.
- Milling TJ, Rose J, Briggs WM, Birkhahn R, et al. Randomized, controlled clinical trial of point-of-care limited ultrasonography assistance of central venous cannulation: the Third Sonography Outcomes Assessment Program (SOAP-3) Trial. *Crit Care Med* 2005;33:1764-1749.
- Preas HL, Suffredini AF. Pulmonary artery catheterization: insertion and quality control. In Tobin MJ, ed. *Principles and Practice of Intensive Care Monitoring*. New York, NY: McGraw-Hill; 1998, 773-795.
- Slama M, Novara A, Safavian A, Ossart M, et al. Improvement of internal jugular vein cannulation using an ultrasound-guided technique. *Intensive Care Med* 1997;23:916-919.
- Turnage WS, Fontanet H. Transesophageal echocardiography-guided pulmonary artery catheter placement. *Anesth Analg* 1993;77:858-859.

RATIONALE (12)

Answer: B

Accuracy of measurement is essential to invasive and noninvasive pressure methodology. Knowledge of pressure transducers is a fundamental component of critical care. The transducer is conventionally placed at the level of the heart in most patients. A change in position of the transducer in relation to the patient is the most common error of pressure measurement. Falsely elevated or lower pressures are observed when the transducer is located below or above the level of the heart, respectively. It is not necessary to zero a transducer when the height of a patient changes, because there has been no change in atmospheric pressure. The "pop" test is used to examine the dynamic response of the system. It is done by a quick pull and release of the transducer's flush valve. This produces a square wave that reverberates two or three times before returning to a normal pressure wave. A poor response to this indicates overdamping that may be caused by air bubbles, partially closed stop cocks, or a clot on the tip of the catheter. However, the pop test is not needed each time there is a change in position. There is no need to check the pulmonary artery occlusion pressure at the new height unless it is clinically indicated.

#### REFERENCES (12)

Bigatello LM, Schmidt U. Arterial blood pressure monitoring. Minerva Anestesiol 2003;69:201-209.
Fessler HE, Shade D. Measurement of vascular pressure. In Tobin MJ, ed. Principles and Practice of Intensive Care Monitoring. New York, NY: McGraw-Hill; 1998, 91-106
Pittman JA, Ping JS, Mark JB. Arterial and central venous pressure monitoring. Intl Anesth Clin 2004;42:13-30.

#### **RATIONALE (13-16)**

Answers: 13-B; 14-A; 15-D; 16-C

Several measured parameters must be linked to knowledge of cardiovascular physiology to assist with diagnoses aided by the pulmonary artery catheter. A normal cardiac index (CI) =  $2.5-4.2 \text{ L/m}^2/\text{min}$ .

Patients with biventricular failure (C) will have reduced CI and stroke volume (SV) with concomitant rises in central venous pressures (CVP) secondary to right-sided failure and pulmonary artery occlusion pressures (PAOP) secondary to failure of the left ventricle.

During hypovolemia, with normal cardiac function, all of the above values will be low (A).  $SV = CO (LPM)/HR \times 1,000$ .

Hyperdynamic cardiac function is often manifested in hepatic failure with markedly increased CI and SV (E).

Elevation of the CVP (equivalent to elevated jugular venous distention on physical examination) without an equivalent increase in PAOP would be manifested in right heart failure (D).

#### **REFERENCES (13-16)**

Davidson CJ, Bonow RO. Cardiac catheterization. In Braunwald E, Zipes DP, Libby P., eds. *Heart Disease*. Vol. 1. Philadelphia, PA: WB Saunders; 2005, 359-386.

Pinsky MR. Hemodynamic profile interpretation. In Tobin MJ, ed. *Principles and Practice of Intensive Care Monitoring*. New York, NY: McGraw-Hill; 1998, 871-888.

Vender JS, Szokol JW. Hemodynamic assessment in the critically ill patient. In Murray MJ, Coursin DB, Pearl RG, Prough DS, eds. *Critical Care Medicine*. Philadelphia, PA: Lippincott; 2002, 122-136.

RATIONALE (17)

Answer: A

The introduction of commercially available pulse oximeters in the 1980s facilitated the recognition of hypoxemic events that were often overlooked. In patients such as the one described in this question, it has allowed monitoring of oxygenation without the need for arterial blood gas sampling. Pulse oximeters measure oxygen saturation (Sao<sub>2</sub>) that is physiologically related Pao<sub>2</sub> based on the sigmoidal oxyhemoglobin dissociation curve. It is essential to understand the relationship between Sao<sub>2</sub> and Pao<sub>2</sub> and the limitations of pulse oximetry. A 90% Sao<sub>2</sub> is a Pao<sub>2</sub> 60 mm Hg. This value is at the top of the upward steep segment of the oxygen dissociation curve. Values above this, on the horizontal portion of the curve are insensitive to changes in PaO<sub>2</sub>. A SaO<sub>2</sub> 96% could represent a Pao<sub>2</sub> between 60 mm Hg and 160 mm Hg (Sao<sub>2</sub>). Below Sao<sub>2</sub> 90%, the Pao<sub>2</sub> decreases precipitously. Pulse oximeters are inaccurate below Sao<sub>2</sub> 80%.

#### **REFERENCES (17)**

Hanning CD, Alexander-Williams JM. Pulse oximetry: a practical review. *Br Med J* 1995;11:367-370. Jubran A. Pulse Oximetry. In Tobin MJ, ed. *Principles and Practice of Intensive Care Monitoring*. New York, NY: McGraw-Hill; 1998, 261-287.

Tobin MJ. Respiratory monitoring. JAMA 1990;264:244-251.

RATIONALE (18)

Answer: C

This patient arrived in stable condition with the exception that her BP was lower than what was observed in the operating room. The fluid replacement for this procedure was appropriate. Transfusion based on the intraoperative hemoglobin level is not warranted at this time nor is the use of a pressor to increase her BP. Her BP at this time can be monitored adequately with noninvasive methods. The size of the BP cuff in relation to the dimension of the patient's arm will affect the measured values. BP cuffs that are too large in either width or length will underestimate the true BP. Similarly, overestimation will occur with cuffs that are too small. Noninvasive pressure should be checked in both arms since there is often a discrepancy in elderly patients. The use of an appropriate-sized BP cuff may be the only intervention needed in this patient.

#### **REFERENCES (18)**

- Lodato RF. Arterial pressure monitoring. In Tobin MJ, ed. *Principles and Practice of Intensive Care Monitoring*. Tobin MJ, ed. New York, NY: McGraw-Hill; 1998, 733-748.
- Singer AJ, Hollander JE. Blood pressure: assessment of interarm differences. *Arch Intern Med* 1996;156: 2005-2008.
- Spiess BD, Gomez MN. Hemodynamic Monitoring. In Longnecker DE, Tinker JH, Morgan GE, eds. *Principles and Practice of Anesthesiology*. St. Louis, MO: Mosby-Year Book; 1998, 802-828.

RATIONALE (19)

Answer: A

Cerebral perfusion pressure (CPP) is defined as the mean arterial blood pressure (MABP) minus the intracerebral pressure (ICP) or jugular venous pressure (JVP) if it is higher. Maintenance of CPP in brain-injured patients is critical because the autoregulation of normal cerebral blood flow between 50 mm Hg and 150 mm Hg may be altered. The arterial transducer should be placed at the approximate level of the circle of Willis since the head of the bed is elevated. The actual pressure of the cerebral circulation would be lower than that measured at the level of the heart. Cerebral pressure monitoring is needed to calculated the CPP. Transducers do not have to be zeroed when a change in height is made since the atmospheric pressure is unchanged.

#### **REFERENCES (19)**

- Feinstein AJ, Patel MB, Sanui M, Cohn SM, et al. Resuscitation with pressors after traumatic brain injury. J Am Coll Surg 2005;201:536-545.
- Foster N, Engelhard K. Managing elevated intracranial pressure. Curr Opin Anaesthesiol 2004;17:371-376.
- Hlatky R, Furuya, Y, Valadka AB, Robertson CS. Management of cerebral perfusion pressure. Semin Respir Crit Care Med 2001;22:3-12.
- Mulvey JM, Dorsch NW, Mudaliar Y, Lang EW. Multimodality monitoring in severe traumatic brain injury: the role of brain tissue oxygenation monitoring. *Neurocrit Care* 2004;1:391-402.
- Ng I, Lim J, Wong HB. Effects of head posture on cerebral hemodynamics: its influences on intracranial pressure, cerebral perfusion pressure, and cerebral oxygenation. *Neurosurgery* 2004;54:593-597.

RATIONALE (20)

Answer: A

The hemodynamic effects of dopamine administration depend upon the dose of dopamine that is given to the patient. Dopamine primarily acts on splanchnic bed receptors in low doses ( $<5 \,\mu g/kg/min$ ). With doses above this (in this case  $8 \,\mu g/kg/min$ ) the dopamine administration will also have an effect on  $\beta_1$ -adrenergic receptors. In addition to the increase in heart rate, the myocardial performance is also affected and a rise in cardiac output will be observed. At this dose, there is likely to be a small rise in the blood pressure as well.

RATIONALE (21)

Answer: B

Dopamine administration in this range (in this case  $25 \,\mu g/kg/min$ ) will affect  $\alpha$ -adrenergic, receptors as well as  $\beta$ -adrenergic receptors. This would result in vasoconstriction and an increase in systemic vascular resistance. Cardiac output would also be augmented and an expected rise in systolic blood pressure would be seen, as well. At this dose of dopamine administration, a greater rise in systolic BP would be expected compared with the "inotropic dose."

RATIONALE (22)

Answer: E

Phenylephrine is a vasoconstrictor that has  $\alpha$ -adrenergic properties only. Therefore, there would be no increase in heart rate expected. Although a sharp rise in mean arterial pressure can be expected, this may cause either no change or potentially a decrease in cardiac output (in addition to a reflex bradycardia).

RATIONALE (23)

Answer: D

Norepinephrine is a vasoactive agent that will stimulate both  $\alpha$ - and  $\beta$ -adrenergic receptors. A very mild increase or no change in cardiac output is likely to be observed. The potent vasoconstriction will cause an overall increase in mean arterial pressure. A minimal effect on heart rate can be expected. Norepinephrine is a potent agent that can achieve targets for mean arterial pressure without inducing extreme tachycardia that can be detrimental but preserving cardiac output as well.

RATIONALE (24)

Answer: C

Dobutamine is a pure inotrope. It has no vasoconstrictor effects and will not be expected to raise blood pressure. In fact, it can be associated with mild vasodilation, which may actually lower the blood pressure. In this scenario, dobutamine should only be administered in concert with another agent to support the blood pressure. Dobutamine will have positive chronotropic effects, as well. Dobutamine is typically used in patients with cardiogenic shock, but may also be of use in the patient with reversible myocardial depression, as in this case of severe sepsis. See table below.

AGENT	TYPICAL DOSE	CHRONOTROPIC EFFECTS	INOTROPIC EFFECTS	VASOCONSTRICTION
DOPAMINE	6-20 µg/kg/min	++	++	+ or ++ (dose-dependent)
EPINEPHRINE	1-10 μg/min	++	1+	++
NOREPINEPHRINE	2-30 μg/min	+	+	++
PHENYLEPHRINE	20-200 μg/min	-		++
VASOPRESSIN	0.0104 U/min	-	-	++

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#### **REFERENCES (20-24)**

- Chernow B, Roch BL. Pharmacologic manipulation of the peripheral vasculature in shock: Clinical and experimental approaches. *Circulatory Shock*. 1986; 18:141-155.
- Desjars P, Pinaud M, Tasseau F, et al. A reappraisal of norepinephrine therapy in human septic shock. Crit Care Med. 1987; 15:134-137.
- Flancbaum L, Dick M, Dasta J, et al: A dose-response study of phenylephrine in critically ill, septic surgical patients. Eur J Clin Pharmacol. 1997; 51:461-456.
- Gillespie TA, Ambos HD, Sobel BE, Robert R. Effects of dobutamine in patients with acute myocardial infarction. *Am J Cardiol.* 1997; 39:588-594.
- Hannemann L, Reinhart K, Grenzer O, et al. Comparison of dopamine to dobutamine and norepinephrine for oxygen delivery and uptake in septic shock. *Crit Care med* 1995; 23:1962-1970.
- Hollenberg SM, Kavinsky CJ, Parrillo JE. Cardiogenic shock. Ann Intern Med. 1999; 131:47-59.
- Holzer J, Karliner JS, O'Rourke RA, et al. Effectiveness of dopamine in patients with cardiogenic shock. Am J Cardiol. 1973; 32:79-84.
- Martin C, Saux P, Eon B, et al. Septic shock: A goal-directed therapy using volume loading, dobutamine and/or norepinephrine. *Acta Anaesthesiol Scand*. 1990; 34:413-417.
- Redl-Wenzl EM, Armbruster C, Edelmann G, et al. The effects of norepinephrine on hemodynamics and renal function in severe septic shock states. *Intensive Care Med.* 1993; 10:151-154.
- Regnier B, Safran D, Carlet J, et al. Comparative haemodynamic effects of dopamine and dobutamine in septic shock. *Intensive Care Med.* 1979; 5:115-120.

RATIONALE (25)

Answer: C

Although it is controversial as to whether or not deliberate increases in oxygen delivery targeting arbitrary supraphysiologic goals are necessary or even beneficial in critically ill patients, this patient with evidence of hypoperfusion in septic shock would benefit from hemodynamic optimization including augmentation of oxygen delivery, especially in the acute resuscitation phase of therapy. Although the precise impact on oxygen delivery from a given intervention would need to be calculated from the formulas below, there is a more simple method to weigh the magnitude of effect of two different interventions if we just examine the proportions that each of the possible interventions would have relative to each other. This patient's oxygen saturation is already 97%, and because dissolved oxygen in the blood has little impact on oxygen content, raising the arterial saturation would be expected to have little impact on oxygen delivery. An increase of the hemoglobin concentration from 8 to 10 g/dL would represent a 20% increase, and an increase of the cardiac output from 1.5 L/min to 3.0 L/min would represent a 100% increase. Raising the cardiac output, therefore, would have a greater impact on oxygen delivery. Administering additional fluids likely will not augment oxygen delivery very much, as the ability of IV fluids to increase the oxygen delivery is limited to the capacity to increase cardiac output. The equations for oxygen content (CaO<sub>2</sub>) and oxygen delivery (DO<sub>2</sub>) are listed below:

```
CaO_2 = PaO_2 \times 0.003 + (Hb g/dL \times SaO_2 \times 1.34 mL/g)

DO_2 = (PaO_2 \times 0.003 + [Hb g/dL \times SaO_2 \times 1.34 mL/g]) \times CO L/min \times 10 dL/L
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#### **REFERENCES (25)**

Boldt J. Clinical review: Hemodynamic monitoring in the intensive care unit. *Crit Care Clin.* 1996; 12:1031-1042.

Hinds C, Watson D. Manipulating hemodynamic and oxygen transport in critically ill patients. *N Engl J Med.* 1995; 333:1074-1075.

Pinsky MR. Functional hemodynamic monitoring. Intensive Care Med. 2002; 28:386-388.

Rivers EP, Ander DS, Powell D. Central venous oxygen saturation monitoring in the critically ill patient. *Current Opin Crit Care*. 2001; 7:204-211.

Yu M, Levy MM, Smith, F, et al. Effect of maximizing oxygen delivery on morbidity and mortality rates in critically ill patients: A prospective, randomized, controlled study. *Crit Care Med.* 1993; 21:830-838.

RATIONALE (26)

Answer: C

Although filling pressures (both central venous pressure and pulmonary capillary wedge pressure) are commonly used to guide fluid therapy in the case of trying to optimize preload, the precise relationship between these filling pressures and ventricular end-diastolic volumes is not the same for every patient. In fact, in both acutely ill patients and healthy volunteers alike, both central venous pressure and pulmonary capillary wedge pressure have been demonstrated to be poor predictors of ventricular preload. A volumetric assessment is likely much more meaningful in terms of optimizing preload. Fluids could be administered until the cardiac output fails to increase with additional volume loading.

Along these lines, there is no accurate way to predict what the "optimal wedge" will be for a given patient, especially one with a history of cor pulmonale. High numbers for filling pressures do not exclude the possibility of further preload responsiveness. Similarly, other patients may not be preload dependent at numbers that are considered moderately low if the patient's Starling curve is relatively flat. Answer D is incorrect because the pulmonary capillary wedge pressure is thought to be a better indicator of left ventricular preload than central venous pressure, although both have their limitations.

#### **REFERENCE (26)**

Kumar A, Anel R, Bunnell E, et al. Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects. *Crit Care Med* 2004;32:691-699.

RATIONALE (27)

Answer: C

Proper positioning of the pulmonary artery catheter is necessary for accurate measurement and interpretation of pressures. The relationship of the different measured pressures is important to evaluate. The PAOP should be less than or equal to the pulmonary artery diastolic pressure unless there are cardiac abnormalities (i.e. mitral stenosis) or the catheter is in lung zone 1 or 2 and the measured pressure may represent alveolar pressure. If the catheter advances to lung zone 3 it is usually below the left atrium on chest radiograph. Blood should be able to be withdrawn freely unless it is inserted too far peripherally. If correctly positioned, the saturation of blood withdrawn from the catheter tip with the balloon up should be equal to or greater than systemic arterial saturation.

#### **REFERENCES (27)**

Pulmonary artery catheter education program. Available at www.pacep.org. Accessed June 13, 2007. Weiniger CF, Ginosar Y, Sprung C, et al. Arterial and pulmonary artery catheters. In Parrillo JE, Dellinger RP, eds. Critical Care Medicine: Principles of Diagnosis and Management in the Adult. 2nd ed. St. Louis, MO: Mosby; 2001, 36-63.

RATIONALE (28)

Answer: B

This figure in this question depicts an arterial pressure waveform and an airway pressure waveform. There is a decrease in systolic blood pressure after the ventilator positive pressure breath. In patients requiring mechanical ventilation, changes in arterial pressure during mechanical breath may serve as an indicator of underlying hypovolemia. The effects of increased intrathoracic pressure on ventricular filling are accentuated in preload-deficient states, resulting in significant decreases in systolic arterial pressure and widening of the arterial pulse pressure. When these changes are present, they predict fluid responsiveness in patients with hemodynamic instability. In order for these measurements to be utilized, patients should have minimal or absence respiratory effort which may necessitate the use of neuromuscular blocking agents. Systolic pressure variation has been proposed as a useful hemodynamic tool to identify patients who might require further IV fluid loading.

#### **REFERENCES (28)**

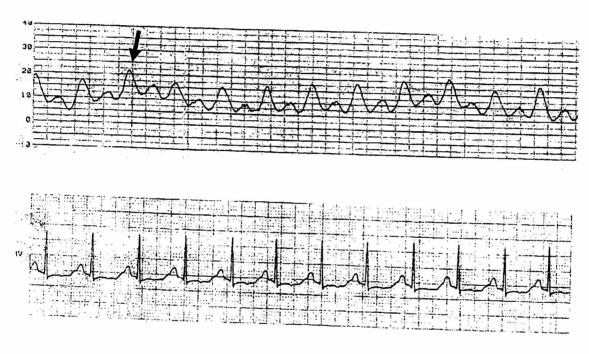
- Lamia B, Chemla D, Richard C, et al. Clinical review: Interpretation of arterial pressure wave in shock states. *Crit Care Med* 2005;9:601-606.
- Michard F, Boussat S, Chemla D, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. *Am J Respir Crit Care Med* 2000;162:134-138.
- Tavernier B, Makhotine O, Lebuffe G, et al. Systolic pressure variation as a guide to fluid therapy in patients with sepsis-induced hypotension. *Anesthesiology* 1998;89:1313-1321.
- Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. *Chest* 2002;121:2000-2008.

RATIONALE (29)

Answer: A

The a-(atrial) wave is associated with atrial contraction and follows the p-wave on the ECG. Some delay is required for the electrical transmission, the indication of contraction, and the transmission of the pulsatile pressure wave to the point of measurement.

In the central venous pressure (CVP) waveform, it is slightly after the p-wave. In the pulmonary artery occlusion pressure (PAOP) tracing, it usually follows the QRS. The v-wave (ventricular) on the CVP and PAOP tracing follows the t-wave (sooner on the former). The arrow points to the v-wave, which in the presence of a normal heart is larger than the a-wave. The PAOP is measured half way down the ascending portion of the a-wave at end expiration. In a mechanically ventilated patient, the end-expiration is the valley portion of the PAOP tracing, which corresponds to the 5th and 6th a-waves on the tracing. Half way down the descending portion of these waves would be approximately 8 mm Hg.



#### **REFERENCES (29)**

Bigatello LM, George E. Hemodynamic monitoring. *Minerva Anestesiol* 2002;68:219-225. Boldt J. Clinical review; hemodynamic monitoring in the intensive care unit. *Crit Care* 2002;6:52-59. Pinsky MR, Payen D. Functional hemodynamic monitoring. *Crit Care* 2005;9:566-572. Pinsky MR. Hemodynamic monitoring in the intensive care unit. *Clin Chest Med* 2003;24:549-560.

# SECTION 7: Infectious Diseases/Immune Dysfunction

# SECTION 7: INFECTIOUS DISEASES/IMMUNE DYSFUNCTION

Instructions: For each question, select the most correct answer.

- 1. An adult patient with no significant past medical history (and never hospitalized) comes to the emergency department because of a soft tissue infection of the leg with purulent drainage. She is admitted to the hospital for antibiotic therapy. Which of the following statements is most correct?
  - A. Methicillin-resistant *Staphylococcus aureus* would be an unlikely pathogen for this community-acquired infection
  - B. Contact precautions are unnecessary
  - C. Monotherapy with cefazolin should be started empirically until culture results are available
  - D. History of close contact with another person having a purulent soft tissue infection has important implications for empiric therapy
- 2. In developed countries, which of the following statements regarding the epidemiology and/or prognosis of critical illness in patients with human immunodeficiency virus (HIV) infection is most correct?
  - A. The incidence of pneumocystis pneumonia as an etiology of respiratory failure is increasing
  - B. Survivors of mechanical ventilatory support for pneumocystis pneumonia rarely live >1 year
  - C. Deaths due to complications of hepatic failure are increasing
  - D. Critically ill patients with HIV have worse short-term clinical outcomes than non-infected patients with a comparable severity of illness

- 3. Which of the following statements about the potential critical care implications of highly active antiretroviral therapy (HAART) in HIV-infected patients is most correct?
  - A. After initiating HAART therapy, acute hepatitis is the most common manifestation of immune reconstitution inflammatory syndrome (IRIS)
  - B. HAART therapy may cause a hyperchloremic metabolic acidosis
  - C. A life-threatening hypersensitivity reaction would most likely be attributed to the use of indinavir in the HAART regimen
  - D. In the absence of suspected HAART toxicity, chronic HAART therapy should be continued when an HIV-infected patient is admitted to the ICU
- 4. Which of the following statements regarding infections in patients receiving hematopoietic stem cell transplants is most correct?
  - A. Pneumonitis due to cytomegalovirus infection is typically an early finding, occurring 0-7 days posttransplant
  - B. Bacterial infections are no longer the main cause of severe infectious complications in the immediate posttransplant period (0-30 days)
  - C. The duration of posttransplant neutropenia has a major impact on the risk of invasive pulmonary aspergillosis
  - D. Pneumocystis pneumonia is a common posttransplant complication even when prophylaxis is administered
- 5. In critically ill surgical patients, which of the following is *not* associated with an increased risk of developing a fungal infection?
  - A. Fungal colonization burden
  - B. Gastrointestinal surgery
  - C. Urinary catheter
  - D. Broad-spectrum antibiotics
  - E. Hemodialysis

- 6. Which one of the following in *not* advocated as a strategy for decreasing antimicrobial resistance in the ICU?
  - A. Formulary restrictions that strictly limit the number of broad-spectrum antibiotics available
  - B. Antibiotic cycling
  - C. Automated antimicrobial selection protocols
  - D. De-escalation (narrowing) of antibiotic regimen as soon as possible after initial empiric therapy
- 7. A patient who has been in the ICU for 2 weeks with a prolonged illness develops a Gramnegative bacteremia from a urinary source. Intravenous ceftriaxone was started when the patient developed fever. Blood culture results are positive for *Escherichia coli*. The isolate is sensitive to ceftriaxone, cefotaxime, and imipenem, but it is resistant to ceftazadime and aztreonam.

On day 2 of the febrile illness, the patient continues to have fever and tachycardia. Which of the following is the best treatment strategy?

- A. Add gentamicin
- B. Order a CT scan to check for a urinary tract obstruction and/or abscess
- C. Repeat the blood culture tests, and add fluroquinolone to the regimen
- D. Discontinue ceftriaxone, and start meropenem
- 8. A 41-year-old male is evaluated for a complaint of sore throat, pleuritic chest pain, and redness and swelling of the anterior neck. He was started on a fluoroquinoline 2 days ago.

Vital signs are as follows:

BP 162/101 mm Hg

HR 110/min

RR 32/min

Temperature 101.2°F

Pharynx examination: bilateral redness with minimal exudate

Chest examination: bilateral basilar crackles

Anterior neck examination: redness, tenderness and swelling, as shown in Figure 1

ECG shown in Figure 2

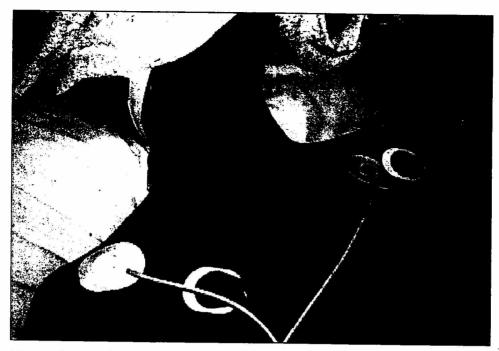


Figure 1. Anterior neck examination.

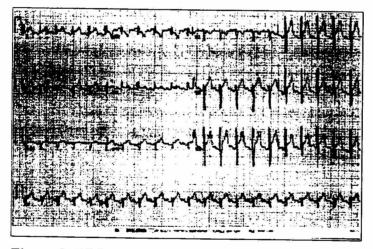


Figure 2. ECG

Which one of the following is most correct concerning this patient's management?

- A. The most important initial step is to discontinue the fluoroquinoline
- B. The patient will require emergent surgical intervention
- C. Creatine kinase will likely be markedly elevated
- D. Rapid resolution of patient symptoms will likely occur with the initiation of antibiotics targeting anaerobic coverage

9. A 52-year-old female with diabetes develops a perirectal abscess, which is drained. Postdrainage, she is placed on clindamycin. She is admitted to the ICU 36 hours later, complaining of severe perineal pain. She is confused, febrile, and hypotensive. Examination of the perineal area reveals edema and redness extending to the inner thighs. Her glucose is 400 mg/dL.

Which one of the following is most appropriate as definitive therapy?

- A. Addition of Gram-negative antibiotic coverage
- B. Hyperbaric oxygenation
- C. Surgical exploration
- D. Insulin therapy for tight glycemic control
- 10. A 65-year-old man is treated for diverticulitis with IV amoxicillin/sulbactam for 5 days. On day 5, he developed a fever and an altered mental status. A chest radiograph revealed a right lower lobe infiltrate. He was intubated, and the sputum culture specimen grew resistant *Pseudomonas*. His antibiotics were changed to ticarcillin/clavulanate and tobramycin.

Two days after the antibiotic change, the patient's fever decreased, and he was more alert. On day 4 after the change of antibiotics, the patient developed a fever to 38.9°C (102.0°F); his white blood cell count increased from 12,000 to 19,000/mm³; and he had several episodes of watery diarrhea. A stool sample was positive for fecal leukocytes and the presence of yeast. Additional medications included IV famotidine, prophylactic low-molecular weight heparin, and enteral feeding with an iso-osmotic nutritional product.

Which one of the following is the most appropriate intervention?

- A. Administer oral metronidazole
- B. Administer IV vancomycin
- C. Administer IV fluconazole
- D. Discontinue enteral feedings
- E. Discontinue ticarcillin/clavulanate

- 11. Skin antiseptics with which of the following will result in the lowest incidence of catheter-related blood stream infections?
  - A. Povidone-iodine
  - B. Antibiotic ointment
  - C. Alcohol
  - D. Chlorhexidine gluconate
- 12. Which of the following patients with sepsis is the most appropriate candidate for treatment with recombinant human activated protein C?
  - A. 60-year-old female with abdominal sepsis on mechanical ventilation 24 hours postlaparotomy, with surgical repair of a transverse colon perforation; the patient is receiving antibiotics and IV fluids, has stable vital signs, and a urine output greater than 1 mL/kg/h
  - B. 55-year-old male with severe community-acquired pneumonia, receiving mechanical ventilation, and requiring norepinephrine for persistent hypotension despite aggressive IV fluids; the patient has a medical history significant for a hemorrhagic stroke within the last 2 months
  - C. 22-year-old female with pyelonephritis and a BP of 110/60 mm Hg after 2 L of IV fluid; laboratory data are significant for a white blood count of 18,000/mL, creatinine of 2.0 mg/dL, normal partial thromblastin time, prothrombin time, and international normalized ratio
  - D. 48-year-old male with acute respiratory distress syndrome secondary to pneumococcal pneumonia; the patient has new elevation of creatinine; laboratory data reveal an international normalized ratio of 2.0, a platelet count of 80,000/mL, and a white blood cell count of 15,000/mL
- 13. Which of the following is a contraindication to the use of recombinant human activated protein C (rhAPC)?
  - A. Platelet count 45,000/mL
  - B. International normalized ratio of 2.5
  - C. Ischemic stroke within 6 months
  - D. Hemoglobin 7.5 g/dL
  - E. Mass on CT scan of brain

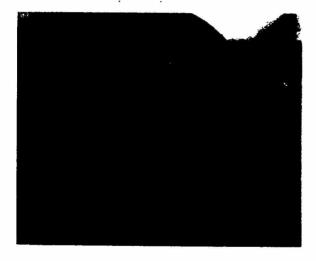
- 14. Which one the following is most correct in regards to Candida-related infections in ICU patients?
  - A. Candiduria should be treated with amphotericin B bladder washings
  - B. Candida parapsilosis fungemia is rarely associated with an indwelling central venous catheters
  - C. Positive blood culture data in a neutropenic patient is not an absolute indication for removal of a tunneled catheter
  - D. Patients receiving parenteral nutrition should receive Candida prophylaxis with fluconazole
- 15. Which of the following patient populations is *least* likely to benefit from fungal prophylaxis with fluconazole?
  - A. Bone marrow transplant patient
  - B. Liver transplant patient
  - C. Patients with persistent gastrointestinal fistula and peritoneal leakage
  - D. Patients receiving parenteral nutrition and broad spectrum antibiotics
- 16. Which of the following patients is most likely to benefit from treatment with IV hydrocortisone (200-300 mg/day)?
  - A. A 58-year-old male admitted to the ICU with acute respiratory distress syndrome and acute pancreatitis receiving mechanical ventilation
  - B. A 35-year-old female presenting to the emergency department with hematemesis and hypotension after a 500 mL bolus of 0.9% normal saline solution
  - C. A 70-year-old male presenting to the emergency department with pneumonia, with the following vital signs: HR 108/min, BP 105/70 mm Hg, and RR 22/min; laboratory data are significant for a lactate of 5.5 mmol/L
  - D. A 45-year-old male admitted to the ICU with Gram-positive bacteremia, and, after 3 L of 0.9% saline solution and several hours of high-dose norepinephrine, it is persistently difficult to maintain a mean arterial pressure >65 mm Hg

- 17. Which of the following statements regarding antiretroviral therapy in critically ill HIV patients admitted to the ICU is most correct?
  - A. Antiretroviral therapy should be stopped while patients are in the ICU
  - B. Discontinuation of antiretroviral therapy increases the risk of patients developing immune reconstitution syndrome
  - C. Antiretroviral drugs should be continued at half the usual dose while patients are in the ICU
  - D. Initiation of antiretroviral therapy in the ICU should be considered for patients admitted with AIDS-defining conditions
- 18. You are asked to evaluate a 48-year-old male who presented to the hospital with a 2-day history of fever, throat pain, and dysphagia. Past medical history is significant for alcohol abuse and hypertension. Vital signs are as follows: temperature 38.2°C, HR 110/min, BP 140/90 mm Hg, and RR 20/min. Pulse oximetry while receiving room air is 100%. On examination, there is a brawny, hard induration of the floor of the oral cavity with an elevation of the tongue. Oral hygiene and dentition are very poor. The oropharingeal mucosa is erythematous, and there is no stridor on auscultation.

In addition to close airway monitoring, which of the following regimens is most appropriate for this case?

- A. Penicillin G + metronidazole
- B. Clindamycin
- C. Levofloxacin
- D. Ceftriaxone + vancomycin
- E. Amphothericin B

- 19. Which one of the following antibiotics is not associated with Clostridium difficile disease?
  - A. Ceftriaxone
  - B. Gatifloxacin
  - C. Clindamycin
  - D. Piperacillin/tazobactam
  - E. Gentamicin
- 20. An elderly diabetic woman undergoes outpatient laparoscopic cholecystectomy for acute cholecystitis. She is sent to the emergency department on the second postoperative day after complaints of severe abdominal pain. Examination reveals altered mental status, high fever, a creatine of 4.2 g/dL (acute), and a lactate of 4.2. Glasgow coma scale score is 10. Family members fear that the patient has suffered a stroke. A photograph of the wound is shown below.



Which one of the following is the most appropriate course of action?

- A. Immediate surgical consult for operative debridement
- B. Oral cephalexin with follow-up in the surgeon's office
- C. Broad-spectrum antibiotics to include anaerobic coverage
- D. Open the wound in the emergency department with later dressing changes

- 21. Which one of the following is most correct concerning central-venous-catheter-associated infection?
- A. The lumen number does not influence the rate of catheter-related complications
- B. Evidence suggests that subclavian catheterization is less likely to result in catheter-related infection than femoral catheterization
- C. Subclavian venous catheterization carries the highest risk of catheter-related thrombosis
- D. Ultrasound guidance offers greater utility for subclavian as compared to internal jugular venous catheterization
- 22. Which one of the following is incorrect concerning the quantitative threshold for diagnosing various ICU infections?
- A. At least 104 organisms/mL in bronchoalveolar lavage for diagnosis of ventilator-associated pneumonia
- B. At least 103 organisms/mL by protected specimen brush for diagnosis of ventilator-associated pneumonia
- C. At least 105 organisms/mL in urinanalysis for urinary tract infection
- D. At least 104 organisms/mL from culture of catheter segment for diagnosis of central-line related infection

# SECTION 7: INFECTIOUS DISEASE/IMMUNE DYSFUNCTION

#### **ANSWERS:**

1-D; 2-C; 3-D; 4-C; 5-C; 6-A; 7-D; 8-B; 9-C; 10-A; 11-D; 12-D; 13-E; 14-C; 15-D; 16-D; 17-D; 18-A; 19-E; 20-A; 21-A; 22-C

RATIONALE (1)

Answer: D

This patient's purulent soft tissue infection should be assumed to be secondary to methicillin-resistant *Staphylococcus aureus* (MRSA) until proven otherwise. In a prospective multicenter study of purulent soft tissue infections in the emergency department setting, Moran and colleagues identified MRSA as the most commonly isolated pathogen. Assuming that this could be MRSA, contact precautions should be used in the hospital.

Monotherapy with cephazolin would not cover MRSA, and, therefore, alternative antibiotics with activity against MRSA should be utilized until culture results are available.

A history of close contact with another person having a purulent soft tissue infection would support the likelihood of MRSA being the pathogen and would certainly necessitate utilization of antibiotics with activity against MRSA.

#### **REFERENCES (1)**

Kollef MH, Micek ST. Methicillin-resistant Staphylococcus aureus: a new community-acquired pathogen? Curr Opin Infect Dis 2006;19:161-168.

Moran GJ, Krishnadasan A, Gorwitz RJ, et al. Methicillin-resistant S. aureus infections among patients in the emergency department. *N Engl J Med* 2006;355:666-674.

Schramm GE, Johnson JA, Doherty JA, Micek ST, Kollef MH. Methicillin-resistant Staphylococcus aureus sterile-site infection: The importance of appropriate initial antimicrobial treatment. *Crit Care Med* 2006;34:2069-2074.

RATIONALE (2)

Answer: C

The incidence of pneumocystis pneumonia as an ideology of respiratory failure in the ICU is beginning to decrease, presumably due to the benefits of highly active antiretroviral therapy (HAART). Contrary to historical data of decades past suggesting that survivors of mechanical ventilation for pneumocystis pneumonia have poor long-term survival, these patients are now experiencing better long-term clinical outcome. This is also likely the effect of HAART.

Critically ill HIV positive patients have similar short-term clinical outcomes compared to HIV negative patients with a comparable severity of illness.

However, complications from liver disease due to co-infection with hepatitis C are now increasing, presumably due to increased long-term HIV-related survival.

#### REFERENCE (2)

Rosen MJ, Narasimhan M. Critical care of immunocompromised patients: human immunodeficiency virus. *Crit Care Med* 2006;34(9 suppl):S245-S250.

RATIONALE (3)

Answer: D

Although the initiation of highly active antiretroviral therapy (HAART) de novo in the ICU setting may not always be indicated when a HIV-infected patient is admitted with acute critical illness, it is generally accepted that (in the absence of suspected HAART toxicity) chronic HAART should be continued in the ICU. Long-term survival is most closely linked with success with antiretroviral therapy.

However, the critical care clinician needs to be aware of several important complications that can be associated with HAART. HAART can cause a severe lactic acidosis. Also, a life-threatening hypersensitivity reaction has been associated with inclusion of abacavir in a HAART regimen. Another possible complication of HAART is the immune reconstitution inflammatory syndrome (IRIS), of which the most common clinical manifestation is acute lung injury.

The lung injury associated with IRIS is believed to be due to systemic inflammation associated with an enhancement of the lymphoproliferative response. As lung injury with IRIS may be difficult to differentiate from an infectious process, the diagnosis of IRIS-mediated lung injury often requires bronchoscopy with bronchoalveolar lavage to exclude an acute infectious process. Patients with IRIS-mediated lung injury improve after discontinuation of HAART.

#### **REFERENCE (3)**

Rosen MJ, Narasimhan M. Critical care of immunocompromised patients: human immunodeficiency virus. Crit Care Med 2006;34(9 suppl):S245-S250.

RATIONALE (4)

Answer: C

Clinicians caring for patients who have received a hematopoietic stem cell transplant must be extremely vigilant about early identification of posttransplant infectious processes. Clearly, these patients are profoundly immunosuppressed. Although the clinician must be concerned with the possibility of either bacterial, fungal, or viral infectious complications at all times, there is a characteristic timeline by which the highest risks for specific infectious agents is most likely to occur.

Bacterial infections remain the main cause of severe infectious complications in the immediate (0-30 day) posttransplant period. The most important viral infection after transplantation is cytomegalovirus (CMV), and CMV pneumonitis is the most severe manifestation of CMV infection. It can manifest with bilateral infiltrates and hypoxemic respiratory failure. The patients at highest risks for CMV infection are those that were CMV seronegative and received stem cells from patients that were CMV seropositive.

Due to the routine use of ganciclovir in high-risk patients immediately following stem cell transplantation, the incidence of CMV disease is decreasing. Acute CMV pneumonitis typically occurs more than 30 days but less than 2 months after stem cell transplantation. The diagnosis is suggested by the presence of diffuse ground glass infiltrates on a high resolution CT scan, and is confirmed by a polymerase chain reaction on fluid from bronchoalveolar lavage or the presence of viral inclusion bodies in lung tissue biopsy. The treatment for CMV disease is ganciclovir. If ganciclovir cannot be tolerated, foscarnet could be used as an alternate agent, but the possibility of acute renal failure is even greater with foscarnet. With effective pneumocystis prophylaxis, the incidence of pneumocystis pneumonia has sharply declined in posttransplant patients.

Invasive pulmonary aspergillosis is a leading cause of severe fungal infection after stem cell transplantation, and can have catastrophic consequences. The onset of invasive pulmonary aspergillosis has a bimodal distribution, with an early peak during the neutropenic phase. The risk of acute-phase pulmonary aspergillosis is a function of the degree and duration of neutropenia associated with transplantation.

#### **REFERENCE (4)**

Soubani AO. Critical care considerations of hematopoietic stem cell transplantation. *Crit Care Med* 2006;34(9 suppl):S251-S267.

RATIONALE (5)

Answer: C

Risks for developing a fungal infection in the ICU are numerous, and include: presence of a central venous catheter, lack of enteral nutrition, utilization of total parenteral nutrition, utilization of broad spectrum antibiotics (with an especially heightened risk with utilization of anaerobic coverage), hemodialysis, gastrointestinal surgery, and any degree of immunosuppression. In addition, colonization with *Candida* species is a major risk factor for developing a fungal infection. Both the duration and the number of sites colonized with *Candida* species have been identified as important risk factors for developing fungal infection.

Although the presence of candiduria may indicate an increased risk of developing fungal blood stream infection, simply having a urinary catheter in place does not.

#### **REFERENCE (5)**

Lipsett PA. Surgical critical care: fungal infections in surgical patients. *Crit Care Med* 2006;34 (9 suppl):S215-S224.

RATIONALE (6)

Answer: A

The indiscriminate use of broad-spectrum antibiotics in the ICU setting can result in increased infections with antimicrobial resistant organisms. Therefore, decreasing broad-spectrum antibiotic use and narrowing the antibiotic regimen to specifically target the identified pathogen as soon as possible after initial empiric therapy are critical actions.

However, critical care clinicians caring for patients with a high severity of illness must often utilize broad-spectrum antibiotics early in the course of therapy, since the consequences of failing to appropriately cover a pathogen could have catastrophic consequences. Evidence-based strategies for decreasing antimicrobial resistance in the ICU setting include antibiotic "cycling" (rotating empiric antibiotic choices at regularly scheduled intervals in order to minimize resistance to any one particular drug) and automated evidence-based protocols for selecting antibiotics.

Over time, heterogeneity in antibiotic utilization helps minimize antimicrobial resistance in the ICU, while homogeneity in antibiotic use clearly increases antimicrobial resistance in ICU pathogens. Therefore, a highly restrictive formulary that limits the number of broad-spectrums antibiotics to a select few would be unfavorable.

#### REFERENCE (6)

Kollef MH, Micek ST. Strategies to prevent antimicrobial resistance in the intensive care unit. *Crit Care Med* 2005;33:1845-1853.

RATIONALE (7)

Answer: D

Despite the susceptibility results, the organism is not actually sensitive to ceftriaxone. Extended-spectrum  $\beta$ -lactamases (ESBLs) are plasmid-mediated enzymes that inactivate all  $\beta$ -lactam antibiotics, except for cephamycins (cefoxitin) and carbapenems (imipenem, meropenem). The organisms may also be resistant to aminoglycosides and fluoroquinolones. The enzymes are found in a variety of Enterobacteriaceae (most commonly *Klebsiella pneumoniae* and *Escherichia coli*). ESBLs are challenging, both because of the difficulty in detecting the presence of ESBLs, and the limited treatment options available.

ESBLs most likely have emerged because of widespread use of third-generation cephalosporins and aztreonam. Because ESBLs are plasmid-mediated, the enzymes are easily transferred to other bacteria species. Risk factors for infection with ESBL-producing organisms include prolonged ICU stay, high severity of illness, long-term antibiotic exposure, and instrumentation and/or indwelling catheters of any kind.

Detection of ESBLs is often difficult. To quantify the extent of the problem in an institution, determining the rate of *in vitro* ceftazadime resistance is one way. The organism may appear to be sensitive to other third-generation cephalosporins *in vitro*. However, the organism will not be clinically susceptible to third-generation cephalosporins, and a treatment failure will result.

For this patient, the *in vitro* susceptibilities included ceftriaxone. At the site of infection, there is a high colony count that would inactivate the antibiotic. The minimum inhibitory concentration at the site of infection would be much higher, and use of ceftriaxone would result in a clinical treatment failure. The tip-off for an ESBL-producing organism was the *in vitro* resistance to ceftazadime. The ESBL E-test (AB Biodisk, Sweden) is the most sensitive method for detecting ESBLs.

Carbapenems (specifically meropenem) are the most effective agents against ESBLs. An ESBL E-test should be performed for this isolate, and the patient should be started on meropenem pending the results.

#### **REFERENCES (7)**

Center for Disease Control. Fact sheet: laboratory detection of extended-spectrum beta-lactamases (ESBLs). Available at: www.cdc.gov/ncidod/dhqp/ar\_lab\_esbl.html. Accessed June 19, 2007.

Nathisuwan S, Burgess DS, Lewin JS. Extended-spectrum beta-lactamases in the 21st century: characterization, epidemiology, and detection of this important resistance threat. *Pharmacotherapy* 2001;21:920-928.

Wiener J, Quinn JP, Bradford PA, et al. Multiple antibiotic-resistant Klebsiella and Escherichia coli in nursing homes. JAMA 1999;281:517-523.

RATIONALE (8)

Answer: B

Clues to the diagnosis in this patient are the previous pharyngeal infection, the redness and swelling of the anterior neck extending to the sternal notch, the pleuritic chest pain, and the ECG finding of pericarditis. The ECG indicates a diffuse ST-segment elevation (or PR-segment depression).

This patient has mediastinitis due to extension of an anterior neck infection due to preexisting pharyngitis. The causative organism in this case was  $\beta$ -hemolytic streptococcus. The CT scan indicates edema in the pretracheal anterior neck area and upper mediastinum associated with the loss of normal tissue planes (see Figure 2 in the question).

Creatine kinase will not likely be markedly elevated in this condition.

Although anaerobic coverage would be indicated, the key is surgical source control. Depending on the fluoroginolone chosen, it may or may not offer adequate empiric coverage for this patient.

#### **REFERENCES (8)**

- Corsten MJ, Shamji FM, Odell PF, et al. Optimal treatment of descending necrotizing mediastinitis. Thorax 1997;52:702-708.
- Kiernan PD, Hernandez A, Byrne WD, et al. Descending cervical mediastinitis. *Ann Thorac Surg* 1998;65;1483-1488.
- Robicsek F. Postoperative sterno-mediastinitis. Am Surg 2000;66:184-192.
- Sakamoto H, Aoki T, Kise Y, et al. Descending necrotizing mediastinitis due to odontogenic infections. Oral Surg Oral Med Oral Pathol Oral Radiol Endodontics 2000;89:412-419.

RATIONALE (9)

Answer: C

This patient likely has a severe necrotizing soft tissue infection that requires radical surgical debridement. The extent of the infection is unknown without surgery. Skin, subcutaneous tissue, fascia, and muscle compartments all may be involved. All dead and infected tissue must be removed. If extensive involvement of the perineum is present, a colostomy may be required.

In diabetic patients, the mortality from this infection exceeds 50%. This infection is a mixed-organism infection with fecal organisms. In addition to surgery, the patient should receive broad-spectrum antibiotics targeted to Gram-negative and anaerobic organisms.

Without surgical removal of the dead tissue, antibiotics are ineffective. The patient also requires concurrent fluid replacement to support the circulation. Control of the glucose is not primary and will not be achieved without control of the infection.

The use of hyperbaric oxygen is controversial. It is thought to inhibit further bacterial invasion but does not eliminate the source of the infection.

#### **REFERENCES (9)**

Elliot DC, Kufera JA, Myers RA. Necrotizing soft tissue infections: risk factors for mortality and strategies for management. *Ann Surg* 1996:224:672-683.

Riseman JA, et al. Hyperbaric oxygen therapy for necrotizing fascitis reduces mortality and the need for debridements. Surgery 1990;108:847-850.

RATIONALE (10)

Answer: A

The mostly likely diagnosis in this patient who has been on multiple antibiotics is *Clostridium difficile* infection. Infection can also manifest several days after discontinuation of antibiotics. The treatment of choice is the administration of oral metronidazole (500 mg 3 times daily or 250 mg 4 times daily).

Although oral vancomycin (125 mg 4 times daily) has similar efficacy, its use is discouraged because of the potential risk of promoting vancomycin-resistant *Enterococci*.

If the oral route is precluded, IV metronidazole is also effective, because a moderate concentration of the drug is attainable in the colon. However, IV vancomycin is not effective.

The decision to treat is usually based on a positive *Clostridium difficile* toxin assay, but treatment should be initiated in high-risk patients prior to obtaining results. This patient has several risk factors, including hospitalization, older age, and several days of treatment with penicillin derivative antibiotics. Treatment is indicated based on the presence of the fever, leukocytosis, the evidence of colitis with fecal leukocytes, and the need to continue the antibiotic for a resistant nosocomial pneumonia.

Ideally, the implicated antibiotic should be discontinued, although this may not always be possible in the critically ill patient. Discontinuation of the antibiotic alone may result in resolution, but is not the sole treatment of choice for critically ill patients.

Finding yeast in the stool is not an indication for treatment and *Candida* would not be expected to cause colitis. There are many other causes of diarrhea in the critically ill patient, including osmotic diarrhea due to nutritional products, the use of drugs containing lactulose or sorbitol, and the use of laxatives or nonsteriodal antiinflammatory drugs. There is no indication that any of these agents are implicated in this patient.

Complications of *Clostridium difficile* infection include toxic megacolon (rare) and a relapse that occurs in 20 to 25% of the cases.

#### **REFERENCES (10)**

Bartlett JG. Antibiotic-associated diarrhea. N Engl J Med 2002;346:334-339.

Guerrant RL. Practice guidelines for the management of infectious diarrhea. *Clin Infec Dis* 2001;32:331-351.

Mylonakis E, Ryan ET, Calderwood SB. Clostridium difficile-associated diarrhea: a review. *Arch Intern Med* 2000;161:525-533.

Recommendations for prevention of the spread of vancomycin resistance: recommendation of the Hospital Infection Control Practices Advisory Committee (HICPAC). MMWR Morb Mortal Wkly Rep 1995;44:1-13.

RATIONALE (11)

Answer: D

Povidone-iodine has been the most widely used antiseptic for cleansing the skin prior to insertion of arterial or central venous catheters. However, a prospective randomized study demonstrated that the preparation of central venous and arterial sites with a 2% chlorhexidine gluconate solution lowered catheter-related bloodstream infections when compared with site preparation with 10% povidone-iodine or 70% alcohol.

A 2% tincture of chlorhexidine for skin antiseptic preparation was approved by the US Food and Drug Administration in 2000. Other preparations of chlorhexidine might not be as effective. In another study, the use of chlorhexidine gluconate 0.5% was no more effective in preventing catheter-related bloodstream infections or central venous catheter colonization than the use of 10% povidone-iodine.

Current recommendations from the Centers for Disease Control and Prevention for decreasing the incidence of catheter-related infections include the use of 2% chlorhexidine gluconate.

#### **REFERENCES (11)**

Humar A, Ostromecki A, Direnfeld J, et al. Prospective randomized trial of 10% povidone-iodine versus 0.5% tincture of chlorhexidine of continuous antisepsis for prevention of central venous catheter infection. *Clin Infect Dis* 2000;31:1001-1007.

Mackie DG, Ringer M, Alvarado CJ. Prospective randomized trial of povidone-iodine, alcohol, and chlorhexidine for prevention of infection associated with central *Lancet* 1991;338:339-343.

RATIONALE (12)

Answer: D

Recombinant human activated protein C (rhAPC), is indicated for patients in severe sepsis at high risk of death. In the PROWESS study, the mortality rates in nearly all the prospectively defined subgroups were lower in the groups that received drotrecogin alfa (activated), when compared with the placebo groups. However, post hoc analysis revealed that the greater the risk of death as determined by an APACHE II score  $\geq 25$ , or the presence of 2 or more organ failures, the greater the benefit in mortality reduction with treatment.

Current recommendations are to consider drotrecogin alfa (activated) for treatment of patients with clinical assessment of high risk of death due to sepsis-induced organ dysfunction, such as an APACHE II score ≥25 or multiple organ failure. If there is no absolute contraindication related to bleeding risks or any relative contraindication that out outweighs the potential benefit of the drug.

Among the small number of patients enrolled in the PROWESS study with single-organ dysfunction and recent surgery (<30 days), all-cause mortality was numerically higher in the drotrecogin alfa (activated) group, when compared with the placebo group. Further analysis of a subgroup of patients with single-organ dysfunction and recent surgery in a separate, randomized, placebo-controlled study (ADDRESS) of septic patients at lower risk of death, revealed that all-cause mortality was also higher in the drotrecogin alfa (activated) group, when compared with placebo. Therefore, it is important to recognize that patients with single-organ dysfunction and recent surgery (<30 days) may not be at high risk of death, irrespective of the APACHE II score, and, therefore, may not be among the indicated population for treatment with rhAPC.

Of the patients presented in the question, the patient illustrated in choice D is the one who best qualifies for rhAPC, as he is at high risk of death and does not demonstrate any contraindications to the drug.

#### **REFERENCES (12)**

Bernard GR, Vincent JL, Laterre PR, et al. Efficacy and safety of recombinant human activated protein C for severe sepsis. N Engl J Med 2001;344:699-709.

Bernard GR. Drotrecogin alfa (activated) recombinant human activated protein C for the treatment of severe sepsis. Crit Care Med 2003; 31(suppl):S85-S93.

Dellinger RP, Carlet JM, Masure H, et al; Surviving Sepsis Campaign Management Guidelines Committee: Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med* 2004; 32:858-873.

Fourrier, F. Recombinant human activated protein C in the treatment of severe sepsis: an evidence-based review. *Crit Care Med* 2004;11:S534-S541.

RATIONALE (13)

Answer: E

The most significant complication for treatment with recombinant human activated protein C (rhAPC) is the potential development of severe bleeding. Of the given choices, the only absolute contraindication to the use of rhAPC is a mass on a CT scan of the brain.

Below is a list of the contraindications and warnings that should be considered when evaluating patients for treatment with rhAPC (drotrecogin alfa [activated]).

#### Contraindications

- 1. Active internal bleeding
- 2. Recent (within 3 months) hemorrhagic stroke
- 3. Recent (within 2 months) intracranial or intraspinal surgery, or severe head trauma
- 4. Trauma with increased risk of life-threatening bleeding
- 5. Presence of an epidural catheter
- 6. Intracranial neoplasm, mass lesion or evidence of cerebral herniation

#### Warnings

- 1. Coagulopathy (platelet count <30,000 mL or international normalized ratio >3.0)
- 2. Concurrent use of heparin
- 3. Recent use of thrombolytics, asprin, IIb-IIIa platelet inhibitors, or other anticoagulants
- 4. Recent (within 6 weeks) gastrointestinal bleed
- 5. Recent (within 3 months) ischemic stroke
- 6. Chronic severe hepatic disease
- 7. Known bleeding diathesis
- 8. Any condition in which bleeding poses as a significant hazard or difficulty to match secondary to its location.

#### **REFERENCES (13)**

Bernard GR. Drotrecogin alfa (activated) recombinant human activated protein C) for the treatment of severe sepsis. Crit Care Med 2003; 31(suppl):S85-S93.

Bernard GR, Vincent JL, Laterre PR, et al. Efficacy and safety of recombinant human activated protein C for severe sepsis. N Engl J Med 2001;344:699-709.

Dellinger RP, Carlet JM, Masure H, et al. Surviving Sepsis Campaign Management Guidelines Committee: Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med* 2004;32:858-873.

Fourrier, F. Recombinant human activated protein C in the treatment of severe sepsis: an evidence-based review. *Crit Care Med* 2004;11:S534-S541.

RATIONALE (14)

Answer: C

Candida-related infections have increased in prevalence over the last several years, particularly in patients hospitalized in the ICU. Current data strongly suggest that candidemia is often related to a central venous catheter. In most patients with positive blood cultures for candida, it is recommended that the central venous be removed. Removal of central venous catheters has been associated with short duration of subsequent candidemia and improved patient outcomes.

This situation may be different for neutropenic patients, particularly those who have lower-risk catheters or long-term catheters, such as Hickman catheters. In patients with neutropenia, edema often results from organisms entering into the blood stream from the gut. In neutropenic patients, it may be difficult to determine whether the candidemia is directly related to an indwelling catheter, such as a Hickman catheter. Therefore, it is reasonable to consider treatment for candidemia in neutropenic patients without removing permanent catheters, unless there is persistent positive blood culture data for more than a few days.

Candida parapsilosis is almost always associated with an central catheter venous.

Asymptomatic candiduria most likely does not benefit from treatment. There are certain situations in which candiduria should be treated, and those include symptomatic candiduria or patients undergoing urologic procedures. In these cases, systemic antifungals are recommended over amphotericin B bladder washings. Amphotericin B bladder washings are only recommended in patients with renal insufficiency and patients who cannot tolerate other antifungals.

Patients receiving parenteral nutrition are not a group that has been identified to benefit from prophylaxis. While patients who received parenteral nutrition are at a higher risk for candidemia, no studies have demonstrated that the use of prophylaxis is advantageous in this particular group.

#### **REFERENCES (14)**

Ortosky-Zeichner L and Rex JH. Antifungal and antiviral therapy. In Dellinger P, Parrillo J, eds. *Critical Care Medicine* 2nd ed. Philadelphia, PA: Mosby; 2001.

Pappas PG, Rex JH, Sobel JD, et al. Guidelines for treatment of candidiasis. Clin Infect Dis 2004;38:161-189.

RATIONALES (15)

Answer: D

Prevention of systemic candidiasis has been explored in different patient populations. The key is to find a patient population with a meaningful rate of invasive candidiasis. The value of prophylaxis has been shown convincingly for bone marrow transplantation patients and select liver transplant patients. Recent reports have also evaluated the use of prophylaxis in ICU patients with persistent gastrointestinal leakage. In this group, the rate of *Candida* peritonitis was very high, and a small, placebo-controlled trial convincingly demonstrated that fluconazole prophylaxis decreased the incidence of candidiasis.

Although patients on parenteral nutrition and broad-spectrum antibiotics are at an increased risk for developing systemic candidemia, no studies have demonstrated that prophylaxis is beneficial in this patient population.

#### **REFERENCES (15)**

Eggimann P, Francioli P, Bille J, et al. Fluconazole prophylaxis prevents intraabdominal candidiosis in high risk surgical patients. *Crit Care Med* 1999;27:1066-1072.

Ortosky-Zeichner L and Rex JH. Antifungal and antiviral therapy. In Dellinger P, Parrillo J, eds. Critical Care Medicine, 2nd ed. Philadelphia, PA: Mosby; 2001.

Pappas PG, Rex JH, Sobel JD, et al. Guidelines for treatment of candidiasis. Clin Infect Dis 2004;38:161-189.

RATIONALE (16)

Answer: D

The use of corticosteroids in critical illness has been the subject of several studies through the years. In the 1980s, efforts concentrated on utilizing cortiscosteroids at very high doses for a short period of time as an antiinflammatory agent in both sepsis and acute respiratory distress syndrome. Multiple, randomized studies failed to demonstrate a benefit of this strategy in critically ill patients. More recently, the use of corticosteroids in patients with septic shock has been revisited. The concept of relative adrenal insufficiency in patients with refractory shock has lead to the rationale of utilizing corticosteroids at physiologic doses for longer periods of time to support adrenal function and improve hemodynamic instability.

Several studies have demonstrated that the use of hydrocortisone in a dose equivalent to 200 mg/day (given as a continuous infusion or in divided doses) improves time to shock reversal and decreases the requirements for vasopressors in patients with septic shock. Annane and collaborators demonstrated in a randomized clinical study that patients who did not respond to the cosyntropin stimulation test (<9 mg/dL) had improved 28-day survival when treated with hydrocortisone (200 mg/day) plus fludrocortisone (25 mg) for 7 days. Patients enrolled in this study were patients who remained hypotensive on vasopressors after appropriate fluid resuscitation.

Current guidelines on the management of patients with severe sepsis and septic shock emphasize the importance of considering this treatment for those patients who remain on vasopressors after fluid resuscitation. Details regarding the appropriate use of corticosteroids in septic shock, such as the need to perform a stimulation test, the need to taper down the dose, and the length of treatment remain unanswered.

Of the patients presented in the question, the patient who best represents the patient group who will most likely benefit from corticosteroids at the given dose is the patient who, after appropriate fluid resuscitation, remains hypotensive on vasopressors. It is very likely that he has relative adrenal insufficiency; hence, the use of corticosteroids may benefit his hemodynamic status and improve his chances of recovery.

#### **REFERENCES (16)**

- Annane D, Bellisaant E, Bollaert PE, et al. Corticosteroids for severe sepsis and septic shock: a systematic review and meta-analysis. *BMJ* 2004;329:480.
- Annane D, Sebille V, Charpentier C, et al. Effect of treatment with low doses of hydrocortisone and fludrocortisone on mortality in patients with septic shock. *JAMA* 2002;288:862-871.
- Dellinger RP, Carlet JM, Masure H, et al. Surviving Sepsis Campaign Management Guidelines Committee: Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med* 2004;32:858-873.
- Minneci PC, Deans, KJ, Banks SM, et al. Meta-analysis: the effect of steroids on survival and shock during sepsis depends on the dose. *Ann Intern Med* 2004;141:47-56.

RATIONALE (17)

Answer: D

The introduction of antiretroviral therapy has dramatically changed the prognosis for patients with HIV infection. As a result, questions regarding the application of antiretroviral therapy in patients with HIV infection in the ICU have arisen. Although several questions remain unanswered, new data have helped elucidate some important aspects of this topic. It is well documented that antiretroviral therapy improves immune function.

Current recommendations for HIV patients admitted to the ICU include, when possible, continuing antiretroviral treatment in those patients who are receiving therapy with evidence of viral load suppression before admission to the ICU. In contrast, for patients with detectable plasma HIV RNA, the benefits of continuing antiretroviral therapy in the ICU are less clear. In these cases, consultation with an HIV expert is recommended, and a case-by-case decision should be made.

The largest subset of patients admitted to the ICU with HIV infections include patients who have not received antiretroviral therapy prior to admission to the ICU. Two studies conducted within the past years suggest that patients who are admitted with an AIDS-defining diagnosis (especially *Pneumocystis* pneumonia) have the poorest prognosis. In theory, treatment with antiretroviral therapy will provide the greatest benefit to these patients. One study evaluated the effects of combination antiretroviral therapy on an age-related illness in the ICU and found a significant improvement in mortality among those patients that received antiretroviral therapy compared to those that did not receive such therapy. On the basis of the available data, it does seem reasonable to initiate antiretroviral therapy in patients admitted to the ICU with an AIDS-defining condition.

#### REFERENCES (17)

- Huang L, Quartin A, Jones D, Havlir D. Intensive care of patients with HIV infection. N Engl J Med 2006;355:173-181.
- Morris A, Creasman J, Turner J, Luce JM, Wachter RM, Huang L. Intensive care of human immunodeficiency virus-infected patients during the era of highly active antiretroviral therapy. *Am J Respir Crit Care Med* 2002;166:262-267.
- Morris A, Wachter RM, Luce J, Turner J, Huang L. Improved survival with highly active antiretroviral therapy in HIV-infected patients with severe *Pneumocystis carinii* pneumonia. *AIDS* 2003;217:73-80.

RATIONALE (18)

Answer: A

The patient is this question presents with clinical features suggestive of Ludwig's angina. Ludwig's angina is a rapidly progressive cellulitis of the floor of the mouth involving the sublingual, submandibular, and submental spaces. Clinically, it is characterized by a brawny discoloration of the floor of the mouth, with an elevation of the tongue; it is very frequently associated airway compromise. The infection is usually caused by decayed teeth and is often seen in patients with a history of alcoholism, diabetes mellitus, and immunodeficiency disorders.

Management is directed toward securing an airway, administering systemic antibiotic therapy, and instituting source control, with early surgical decompression, when needed. The most common causative pathogens are a mixed flora of aerobic and anaerobic bacteria, including streptococcal species, Stapholoccocus aureus, Borrelia vincentii, Fusobacterium, Bacteroides species, and Eikenella corrodens.

Of the possible choices, the most appropriate antibiotic regimen would include pencillin G plus metronidazole to cover the above pathogens. A growing incidence of *Eikenella corrodens*, which does not respond to clindamycin, makes clindamycin monotherapy inadequate.

#### **REFERENCES (18)**

- Ferrara PC, Busino LJ, Snyder HS. Diagnostics: uncommon complications of odontogenic infections. Am J Emerg Med 1996;14:317-327.
- Moreland LW, Corey J, McKenzie R. Ludwig's angina: report of a case and review of the literature. Arch Intern Med 1988;148:461-466.
- Ramadan H, El Solh A. An update on otolaryngology in critical care. Am J Respir Crit Care Med. 2004;169:1273-1277.

RATIONALE (19)

Answer: E

Most antibiotics have been associated with Clostridium difficile disease; the only agents that have not been associated with C difficile are aminoglycosides. Broad-spectrum antibiotics may have a greater effect on normal flora, resulting in C difficile colonization and disease. The risk of disease increases if patients receive multiple agents and have longer courses of therapy. Other risk factors for C difficile disease include older age (>65 years), severe illness, longer hospital stay, and possibly, antiulcer medications.

#### **REFERENCES (19)**

Binardi GE. Risk factors for *Clostridium difficile* infection. *J Hosp Infect* 1998;40:1-5. Loo VG, Poirier L, Miller MA, et al. A predominantly clonal multi-institutional outbreak of *Clostridium difficile*-associated diarrhea with high morbidity and mortality. *N Engl J Med* 2005;353:2442-2449.

Pepin J, Saheb N, Coulombe M-A, et al. Emergence of fluoroquinolones as the predominant risk factor for *Clostridium difficile*-associated diarrhea: a cohort study during an epidemic in Quebec. *Clin Infect Dis* 2005;41:1254-1260.

Sunenshine RH, McDonald LC. Clostridium difficile-associated disease: new challenges from an established pathogen. Cleveland Clin J Med 2006;73:187-197.

RATIONALE (20)

Answer: A

This unfortunate diabetic woman has a rapidly progressing soft tissue infection following laparoscopy and cholecystectomy. The umbilical port is frequently involved, and, as this is the site where the gallbladder was removed through a small opening in the abdominal wall and as the patient has significant subcutaneous fat, contamination is likely. Changes suggestive of progressive soft tissue infection are apparent on the CT scan. Subcutaneous changes, in a patient such as this, can occur without obvious cutaneous manifestations. At the very least, immediate surgical consultation should be obtained.

The patient has a rapidly spreading necrotizing soft tissue infection and shows signs of multisystem organ failure. This patient must be assumed to have deep soft tissue infection extending at least to the fascia and possibly below. Appropriate antibiotic choices include piperacillin-tazobactam and vancomycin. A common offending organism in this setting is streptococci with toxin production. However, staphylococci have recently been shown to cause a toxin syndrome similar to streptococci. This patient received immediate surgical debridement and antibiotics, as described above. The peritoneal cavity was opened and the underlying intestine was uninvolved.

#### **REFERENCES (20)**

- Ahrenholz DH. Necrotizing fasciitis and other soft tissue infections. In: Irwin RS, Rippe JM, eds. *Irwin and Rippe's Intensive Care Medicine*. 5th ed. Philadelphia, PA: Lippincott, Williams & Wilkins; 2003:1709-1716.
- Kravitz GR, Dries DJ, Peterson ML, et al. Purpura fulminans due to Staphylococcus aureus. Clin Infect Dis 2005; 40:941-947.
- McHenry CR, Piotrowski JJ, Petrinic D, et al. Determinants of mortality for necrotizing soft tissue infections. *Ann Surg* 1995; 221:558-565.
- Moran GJ, Krishnadasan A, Gorwitz RJ, et al. Methicillin-resistant *S aureus* infections among patients in the emergency department. *N Engl J Med* 2006; 355:666-674.
- Naimi TS, LeDell KH, Como-Sabetti K, et al. Comparison of community- and health-care-associated methicillin-resistant *Staphylococcus aureus* infection. *JAMA* 2003; 290:2976-2984.

RATIONALE (21)

Answer: A

The lumen number does not directly affect the rate of catheter-related complications. Therefore, the choice of either a single lumen or multilumen catheter should be made according to the type required to deliver the needed medications or nutritional support. Although some evidence suggests that subclavian catheterization may be associated with fewer catheter-related infections than the femoral or internal jugular approaches. However, there has been no head-to-head comparison approaches.

The risk of venous thrombosis is approximately 4 times greater for internal jugular insertion than subclavian venous catheter insertion. Internal jugular catheterization can be difficult in morbidly obese patients in whom the landmarks are often obscured. Ultrasound guidance is particularly useful for internal jugular venous catheterization, and reduces the number of mechanical complication and placement failures, as well as the time required for insertion. Literature support for ultrasound use during subclavian access is less prominent.

#### **REFERENCES (21)**

- Cook D, Randolph A, Kernerman P, et al. Central venous catheter replacement strategies: a systematic review of the literature. *Crit Care Med* 1997;25:1417-1424.
- McGee DC, Gould MK. Preventing complications of central venous catheterization. *N Engl J Med* 2003;348:1123-1133.
- Techgraber UK, Benter T, Gebel M, Manns MP. A sonographically guided technique for central venous access. AJR Am J Roentgenol 1997;169:731-733.
- Timsit JF, Farkas JC, Boyer JM, et al. Central vein catheter-related thrombosis in intensive care patients: incidence, risk factors, and relationship with catheter-related sepsis. *Chest* 1998;1140: 207-213.

RATIONALE (22)

Answer: C

Quantitative techniques for sampling the distal airways by bronchoscopy or nonbronchoscopic (blind) bronchoalveolar lavage utilize at least 104 organisms/mL for diagnosis of ventilator-associated pneumonia. The bronchoscopic technique with the protected specimen brush use 103 organism/mL for cutoff. 105 organisms/mL is routinely used as the threshold for defining urinary tract infection with the Foley catheter culture. For defining catheter-related infection, a measurement of at least 102 organism/mL is recommended.

#### **REFERENCES (22)**

Carven DE, Palladino R, McQuillen DP. Healthcare-associated pneumonia in adults: management principles to improve outcomes. *Infect Dis Clin North Am* 2004;18:939-962.

Centers for Disease Control and Prevention. Available at: www.cdc.gov/. Accessed June 7, 2007. Chastre J, Fagon JY. Ventilator-associated pneumonia. Am J Respir Crit Care Med 2002;165:867-903.

Hospital-acquired Pneumonia Guideline Committee of the American Thoracic Society and Infectious Diseases Society of America: guidelines for the management of adults with hospital-acquired pneumonia, ventilator-associated pneumonia, and healthcare-associated pneumonia. Am J Respir Crit Care Med 2005;171:388-416.

Niederman MS, Torres A, Summer W. Invasive diagnostic testing is not needed routinely to manage suspected ventilator-associated pneumonia. Am J Respir Crit Care Med 1994;150:565-569.

## SECTION 8: Obstetrics/Gynecology

### **SECTION 8: OBSTETRICS/GYNECOLOGY**

<b>Instructions:</b> For each question listed below, select the most correct answer
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1. A 30-year-old pregnant woman at 34 weeks gestation presents with pedal edema and hypertension. Laboratory data include blood urea nitrogen 22 mg/dL, creatinine 1.6 mg/dL, ALT 350 U/L, bilirubin 5 mg/dL, ammonia 70 mg/dL, glucose 50 mg/dL, white blood cell count 19,000/mm³, hemoglobin 9.6 g/dL, platelets 120,000/mm³, prothrombin time 20.2 seconds, and partial thromboplastin time 45 seconds. On physical examination, she is noted to be slightly lethargic, with a few basilar rales bilaterally and a normal size liver on percussion.

Which one of the following is the most likely diagnosis?

- A. Viral hepatitis
- B. Severe pre-eclampsia
- C. HELLP syndrome
- D. Intrahepatic cholestasis of pregnancy
- E. Acute fatty liver of pregnancy

2. A 25-year-old female who is 30 weeks pregnant presents with a severe exacerbation of asthma that began 3 days earlier. Vital signs are BP 100/60 mm Hg, HR 120/min, RR 30/min, and temperature 98.4°F. She is noted to have inspiratory and expiratory wheezing with use of accessory muscles of respiration. Arterial blood gas results while receiving 4 L/min nasal cannula at presentation are pH 7.40, Paco<sub>2</sub> 42 mm Hg, and PaO<sub>3</sub> 68 mm Hg.

Which one of the following is the most appropriate action(s) at this time?

- A. Nebulization of albuterol
- B. Nebulization of albuterol and IV corticosteroids
- C. Subcutaneous terbutaline followed by nebulization of albuterol
- D. Intubation followed by nebulization of albuterol
- E. Noninvasive ventilation with nebulization of albuterol

- 3. Which one of the following is the most correct policy for administration of Rh-immune globulin to pregnant patients with minor abdominal trauma from a motor vehicle accident?
  - A. Rh-negative woman in any stage of pregnancy
  - B. Rh-negative woman in the second trimester of pregnancy
  - C. Rh-negative woman in the third trimester of pregnancy
  - D. Rh-negative woman in any stage of pregnancy with a positive Kleihauer-Betke test
- 4. A 24-year-old woman underwent spinal anaesthesia for fractional dilation and curettage in the lithotomy position. She arrives in the recovery room resting comfortably on 40% oxygen via face mask. She suddenly became agitated, diaphoretic, cyanotic, and developed altered mental status. She was promptly intubated and manually ventilated with 100% oxygen. Physical examination revealed a temperature of 37.8°C (100°F), BP 80/60 mm Hg, HR 44/min, and RR 22/min with bilateral basilar rales and a churning murmur over her left sternal border. Neurologic examination was remarkable for no response to sternal rub with pinpoint pupils and left lateral gaze preference. Arterial blood gases on 100% oxygen revealed pH 7.42, Paco<sub>2</sub> 28 mm Hg, and PaO<sub>2</sub> 68 mm Hg.

Which one of the following would be the most appropriate immediate course of action?

- A. Place the patient in the left lateral decubitus head-down position
- B. Insertion of a central venous catheter for volume replacement
- C. Administer atropine 0.5 mg IV
- D. CT scan of the head
- E. Systemic heparinization
- 5. A 22-year-old female at 36 weeks gestation is hospitalized with premature contractions. She is noted to have BP 169/110 mm Hg, HR 90/min, RR 18/min, and temperature 37.1°C (98.8°F). Urinalysis shows 3+ protein, and she has 1+ edema of her lower extremities. Fetal heart tones are normal. She is started on IV fluids at 200 mL/h, IV magnesium sulfate, and subcutaneous terbutaline. Three hours later, she complains of shortness of breath and cough. While recieving room air, pulse oximetry reveals 91% saturation, RR 26/min, and rales are noted in the lower lung fields. No jugular venous distention or cardiac gallops are appreciated. Chest radiograph is shown on the following page.

Which one of the following is the most likely etiology of the pulmonary edema?

- A. Peripartum cardiomyopathy
- B. Fluid overload
- C. Tocolytic agent
- D. Amniotic fluid embolism
- E. Infection



6. A 30-year-old pregnant woman at 35 weeks gestation presents to the hospital with complaints of headache, nausea, vomiting and weakness. Vital signs are BP 148/90 mm Hg, HR 108/min, RR 18/min, and temperature 37.5°C (99.6°F). She is noted to be lethargic on examination. Laboratory tests show a decrease in hemoglobin from her baseline of 11 g/L to 9.2 g/L, platelet count 70,000/mm³, prothrombin time 12.2 seconds, partial thromboplastin time 35 seconds, blood urea nitrogen 22 mg/dL, creatinine 1.4 mg/dL, ALT 50 U/L, bilirubin 1.0 mg/dL, and glucose 90 mg/dL, Urinalysis showed 2+ protein, trace blood.

Which one of the following is the most appropriate initial intervention intervention?

- A. Delivery of the fetus
- B. IV magnesium sulfate
- C. Plasma exchange
- E. CT scan of head
- 7. An 18-year-old female with no prenatal care delivered vaginally. The next day, she is noted to have a BP of 190/110 mm Hg, pulse 110/min, RR 18/min, afebrile. Her urine output is noted to be 25 mL/h for the last 5 hours. Which one of the following interventions is most appropriate?
  - A. IV furosemide
  - B. IV enalaprilat
  - C. IV magnesium sulfate
  - D. Fluid challenge and observation
  - E. Intubation and mechanical ventilation

8. A 38-year-old female who is 30 weeks pregnant is admitted to the ICU with sepsis from pyelonephritis. The patient is treated with IV antibiotics and volume resuscitation with clinical improvement. One day after admission, the patient develops a cardiac arrest, and a code is called.

Which one of the following statements is most correct?

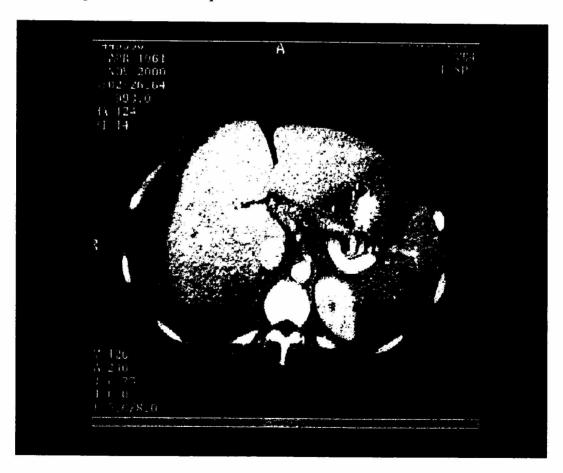
- A. The optimal position for resuscitation is supine, with elevation of lower extremities
- B. The optimal position for resuscitation is left lateral decubitus
- C. Epinephrine is contraindicated in pregnant patients
- D. The right hip should be elevated at a 15 to 30° angle
- E. Failure to achieve a rhythm and viable blood pressure in 5 minutes should prompt emergent cesarean section to salvage fetus
- 9. Which one of the following suggests the diagnosis of amniotic fluid embolism rather venous air embolism?
  - A. Sudden cardiovascular collapse
  - B. Occurrence during labor
  - C. Diffuse bilateral pulmonary infiltrates
  - D. Disseminated intravascular coagulation

10. A 39-year-old female who is 29 weeks pregnant is admitted to the hospital with vaginal bleeding and complaints of right upper quadrant (RUQ) abdominal pain. The patient has a spontaneous abortion secondary to placental abruption. Several hours later, the patient develops hypotension, tachycardia, and complains of increased RUQ pain.

Laboratory data show the following values: sodium 130 mEq/L, potassium 5.1 mEq/L, blood urea nitrogen 34 mg/dL, creatinine 2.1 mg/dL, white blood count 11,000/mm³, hemoglobin 5.9 g/L, hematocrit 17%, platelets 32,000/mm³, international normalized ration 1.1, partial thromboplastin time 26 seconds, total bilirubin 1.2 mg/dL, lactate dyhydrogenase 20,320 U/L, AST 2,340 U/L, ALT 2,100 U/L, and GGT 40 U/mL.

A CT scan of the abdomen was done and is shown below (see figure). The patient was transferred to the ICU. Which one of the following management options is most appropriate at this point?

- A. Supportive therapy with transfusion of packed red blood cells and platelets
- B. Transfuse packed red blood cells and fresh frozen plasma, followed by emergent laparotomy
- C. CT-scan-guided percutaneous drainage
- D. Plasmapheresis
- E. Enroll patient in liver transplant list



11. A 30-year-old woman has been undergoing fertility treatments with ovulation induction with human chorionic gonadotropin (hCG). She is admitted to the hospital with abdominal pain and distension, tachycardia, and respiratory difficulty. Vital signs are BP 110/60 mm Hg supine and 90/52 mm Hg standing, HR 118/min supine and 130/min standing, RR 24/min, and temperature 98.4°F. Physical examination revealed diffuse abdominal tenderness, with a moderate amount of tense ascites. Laboratory is remarkable for hematocrit 45%, white blood cell count 18,000/mm³, and mild elevation of liver transaminases. Pulse oximetry is 96% while receiving room air.

Which one of the following is the most likely diagnosis for this patient?

- A. Ectopic pregnancy
- B. Ruptured ovarian cyst
- C. Pelvic inflammatory disease
- D. Ovarian hyperstimulation syndrome
- 12. Which of the following is an expected (ie, normal) change in cardiopulmonary physiology associated with third trimester pregnancy?
  - A. Increased venous return to the heart in the supine position
  - B. Increased pulmonary artery occlusion pressure
  - C. Decreased total circulating blood volume
  - D. Decreased systemic vascular resistance
  - E. Decreased HR
- 13. Which one of the following statements is true regarding treatment of lower extremity venous thrombosis in a pregnant woman at 30 weeks gestation?
  - A. Unfractionated heparin is superior to low-molecular-weight heparin
  - B. Coumadin is absolutely contraindicated
  - C. Heparin does not need to be continued after delivery
  - D. Twice-daily dosing of low-molecular-weight heparin is preferred
  - E. An inferior vena cava filter should be placed to prevent pulmonary embolism

## SECTION 8: OBSTERTICS/GYNECOLOGY

#### **ANSWERS:**

1-E; 2-D; 3-A; 4-A; 5-C; 6-C; 7-C; 8-D; 9-D; 10-A; 11-D; 12-D; 13-D

RATIONALE (1)

Answer: E

There are several potential etiologies of liver disease in pregnancy and distinguishing between them may be difficult. The laboratory data in this patient are most consistent with acute fatty liver of pregnancy that often presents in a fulminant manner. The table below lists some of the helpful laboratory findings in acute fatty liver, eclampsia/pre-eclampsia, and HELLP syndrome. In acute fatty liver of pregnancy, the liver size is normal or small, and the ALT levels rise to higher levels than in eclampsia/pre-eclampsia or HELLP syndrome. Signs of hepatic failure, such as higher levels of bilirubin, hypoglycemia, elevated ammonia level, and DIC, are often present. Hepatitis in pregnancy will present with similar laboratory findings as a nonpregnant patient with higher levels of transaminases, absence of DIC, and often an enlarged liver. Intrahepatic cholestasis of pregnancy is a benign condition associated with pruritis, jaundice, elevated transaminases, elevated bilirubin, and normal prothrombin time The most difficult distinction in a pregnant patient is liver disease due to eclampsia/severe pre-eclampsia and HELLP syndrome. The ammonia level may be particularly helpful in these situations. Consultation with a maternal fetal specialist is warranted for consideration of immediate delivery.

Table 1. Differentation of liver disease

Test	Acute fatty liver	Eclampsia/pre-eclampsia	HELLP
Liver size	Small	Normal or T	Normal or 1
ALT (usual range)	300 U/L	60 U/L	150 U/L
Bilirubin	<b>↑</b>	Normal, mild ↑	Normal, mild ↑
Glucose	$\downarrow$	Normal	Normal
Ammonia	1	Normal	Normal
DIC	75%	Rare	20-40%

#### REFERENCES (1)

Guntupalli SR, Steingrub J. Hepatic disease and pregnancy: an overview of diagnosis and management. Crit Care Med. 2005; 33:S332-S339.

Rahman TM, Wendon J. Severe hepatic dysfunction in pregnancy. *Q J Med*. 2002; 95:343-357. Wakim-Fleming J, Zein NN. The liver in pregnancy: disease vs benign changes. *Cleveland Clin J Med*. 2005; 72:713-721.

RATIONALE (2)

Answer: D

This patient has severe, stage 4 asthma with  $CO_2$  retention. The normal  $Paco_2$  in a pregnant woman is 30-32 mm Hg, therefore, a  $Paco_2$  of 42 mm Hg represents significant hypercapnia due to airway obstruction. In addition, the patient has impaired oxygenation which needs improvement quickly to ensure oxygen delivery to the fetus. The best option for this patient is intubation followed by aggressive treatment of the bronchospasm and airway inflammation with a nebulized  $\beta_2$ -agonist, such as albuterol and IV administration of corticosteroids. Both agents are safe to administer in pregnancy. Systemic  $\beta$ -agonists have greater side effects, with no added benefit compared with inhaled agents. Noninvasive ventilation could be considered as an option in less severe respiratory conditions but the risk of aspiration may be increased.

#### **REFERENCES (2)**

Graves CR. Acute pulmonary complications during pregnancy. Clin Obstet Gynecol. 2002; 45:369-376.

Hanania NA, Belfort MA. Acute asthma in pregnancy. Crit Care Med. 2005; 33:S319-S324.

RATIONALE (3)

Answer: A

Even minor trauma can be associated with significant fetomaternal hemorrhage. Guidelines suggest that all Rh-negative pregnant women with abdominal trauma should be given anti-D immunoglobulin. Rh antigen is developed fully in fetuses by 6 weeks of gestation, therefore alloimmunization can occur in the first trimester. The Kleihauer-Betke test can quantify fetomaternal hemorrhage greater than 0.5 mL, but maternal exposure to as little as 0.15 mL can elicit immunization. A negative test does not obviate the need for administration of Rh-immune globulin. The Kleihauer-Betke test is important to determine the need for additional dosing based on quantity of fetomaternal hemorrhage.

#### **REFERENCES (3)**

American College of Obstetrics and Gynecology. Prevention of Rh D alloimmunization: clinical management guidelines for obstetrician-gynecologists. *Int J Gynaecol Obstet.* 1999; 66:63-70. Grossman NB. Blunt trauma in pregnancy. *Am Fam Physician.* 2004; 70:1303-1310. Subcommittee on Advanced Trauma Life Support of the American College of Support.

Subcommittee on Advanced Trauma Life Support of the American College of Surgeons. Trauma in women. In: *Advanced Life Support Program for Doctors*. Chicago, IL: American College of Surgeons; 1997: 377-387.

Van Hook JW. Trauma in pregnancy. Clin Obstet Gynecol. 2002; 45:414-424.

RATIONALE (4)

Answer: A

The sudden occurrence of unexplained cardiopulmonary dysfunction with neurologic findings during or soon after an obstetric/gynecologic surgical procedure should suggest the possibility of venous air embolism leading to paradoxic arterial embolism. Whenever a surgical wound disrupts veins, creating a blood-air interface that lies above the level of the right atrium, there is a potential for negative intravascular pressure to create venous air emboli. Venous air embolism may occur during or after a caesarean section, orogenital sex, normal labor with placenta previa, or illegal abortion.

The primary pathophysiologic event in venous embolism is intense vasoconstriction of the pulmonary circulation, which results in ventilation/perfusion mismatch leading to hypoxia, interstitial pulmonary edema, and systemic hypotension leading to reduced cardiac output as pulmonary vascular resistance increases. Paradoxic arterial air embolization may further complicate the course of a patient who has a patent foramen ovale. As venous emboli increase right atrial pressure, a patent foramen ovale may open, thereby creating a right-to-left shunt by which air reaches the arterial circulation. The precordial "mill wheel" murmur is characteristic of venous air embolism. The appropriate management is to place the patient in the left lateral decubitis headdown position to decrease the venous air leaving the pulmonary outflow tract. Oxygen (100%) should be administered to allow reabsorption of nitrogen. If a central venous catheter is in place, aspiration of air can be attempted. Other supportive measures, such as mechanical ventilation, are utilized as indicated. Volume replacement is unlikely to correct the hemodynamics in this patient and may increase right-sided pressure and lead to further paradoxic arterial emboli. Atropine will also have no benefit in this patient. A head CT scan would delay appropriate management, and systemic heparinization is not indicated, because the clinical situation does not suggest pulmonary embolism. Use of hyperbaric oxygenation is controversial.

#### **REFERENCES (4)**

- Gei AF, Vadhera RB, Hankins GDV. Embolism during pregnancy: Thrombus, air, and amniotic fluid. *Anesthesiology Clin N Am.* 2003; 21:165-182.
- Jiva TM. Critical care of pregnant women, Part 2: Fluid and air embolism, pneumonia. *J Crit Illness*. 2000; 15:424-429.
- Stoloff DR, Isenberg RA, Brill AI. Venous air and gas emboli in operative hysteroscopy. *J Am Assoc Gynecol Laparosc.* 2001; 8:181-192.

RATIONALE (5)

Answer: C

Pulmonary edema in pregnancy is associated with an increased risk of maternal and fetal morbidity and mortality. In this patient, the most likely cause of the pulmonary edema is the use of a tocolytic agent, terbutaline. A recent study identified the combination of magnesium sulfate and subcutaneous terbutaline as the most common cause of tocolytic-associated pulmonary edema. The exact pathophysiology is controversial, and potential mechanisms include fluid overload from salt and water retention, increased cardiac output and hydrostatic pressure, and increased capillary permeability. Possible risk factors for tocolytic-associated pulmonary edema include multiple gestations, pre-eclampsia, and sepsis. Therapy includes discontinuation of the agents, supplemental oxygen, and possible diuretic administration. Although peripartum cardiomyopathy may present in the last month of pregnancy, the absence of jugular venous distention or cardiac gallop, as well as the acuteness of onset, make this diagnosis less likely. Fluid overload has also been identified as a cause of pulmonary edema in pregnancy, but the amount of fluid administered in this patient would have only been 600 mL prior to the development of symptoms. Most fluid overload associated with pulmonary edema has occurred in the postpartum period. Other causes of pulmonary edema in a pregnant patient include pre-eclampsia, severe infection, and cardiac disease, such as valvular disorders and hypertrophic subaortic stenosis.

#### **REFERENCES (5)**

Jiva TM. Critical care of pregnant women, part 1: pulmonary edema, ARDS, thromboembolism. *J Crit Illness*. 2000;15:316-324.

Lapinsky SE. Cardiopulmonary complications of pregnancy. *Crit Care Med.* 2005;33:1616-1622. Pisani RJ, Rosenow EC. Pulmonary edema associated with tocolytic therapy. *Ann Intern Med.* 1989; 110:714-718.

Rizk NW, Kalassian KG, Gilligan T, et al. Obstetric complications in pulmonary critical care medicine. *Chest.* 1996;110:791-709.

RATIONALE (6)

Answer: C

The patient most likely has thrombotic thrombocytopenic purpura (TTP), although it may be difficult or impossible at a single point in time to distinguish TTP from HELLP syndrome and severe preeclampsia. TTP is associated with pregnancy. Plasma exchange and corticosteroids would be the treatment of choice. Features of this patient's presentation that are more characteristic of TTP include the presence of lethargy, normal lung function tests, and absence of DIC with prolonged prothrombin time and partial thromboplastin time. Microangiopathic hemolytic anemia would be present in TTP and HELLP syndrome. HELLP syndrome may not be associated with hypertension, but abnormal liver function tests are required for diagnosis. The treatment of choice for HELLP syndrome and severe preeclampsia is delivery. If symptoms and signs fail to resolve after delivery, the diagnosis of TTP may be more likely.

#### **REFERENCES (6)**

- McCrae R. Thrombocytopenia in pregnancy: differential diagnosis, pathogenesis, and management. *Blood Rev.* 2003;17:7-14.
- McMinn JR, George JN. Evaluation of women with clinically suspected thrombotic thrombocytopenic purpura-hemolytic uremic syndrome during pregnancy. *J Clin Apheresis*. 2001;16:202-209.
- Shamseddine A, Chehal A, Usta I, et al. Thrombotic thrombocytopenic purpura and pregnancy: report of four cases and literature review. *J Clin Apheresis*. 2004;19:5-10.

RATIONALE (7)

Answer: C

This patient is most likely suffering from severe pre-eclampsia, which can occur up to 1 week after delivery. The treatment of choice with elevated BP is the administration of magnesium sulfate to prevent progression to eclampsia. The recommended dose is 2 g IV bolus, followed by a 1-2 g/h continuous IV infusion. Further BP control may be needed, if the diastolic blood pressure remains above 100 mm Hg. Because patients with pre-eclampsia/eclampsia are intravascularly volume depleted, diuretics are not indicated. Although a fluid challenge may be appropriate in this patient, continued observation without intervention with magnesium is inappropriate. There is no indication for intubation.

#### **REFERENCES (7)**

- ACOG practice bulletin. Diagnosis and management of preeclampsia and eclampsia. *Int J Gynecol Obstet*. 2002;77:67-75.
- Chames MC, Livingston JC, Ivester TS, et al. Late postpartum eclampsia: a preventable disease? Am J Obstet Gynecol. 2002;186:1174-1177.
- Lain KY, Roberts JM. Contemporary concepts of the pathogenesis and management of preeclampsia. *JAMA*. 2002; 287:3183-3186.
- Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. *Obstet Gynecol*. 2003;102:181-192.

RATIONALE (8)

Answer: D

Cardiopulmonary arrest during pregnancy is treated with the same advanced cardiac life support protocols as in nonpregnant patients. The only difference is the optimal recommended position for resuscitation. During pregnancy, the gravid uterus compresses the inferior vena cava, limiting venous return to the heart. This compression increases with the size of the uterus and varies with the patient's position. Compression is maximal in the supine and right decubitus positions and is minimized with the left lateral decubitus. Blood flow and hemodynamics will improve with left lateral ducubitus position, but efficacy to provide chest compressions should also be considered. Rees and collaborators examined the efficacy of resuscitation with the body at various angles of inclination. At 0° (supine position) the efficacy of chest compressions is maximal, whereas at 90°, chest compressions are least effective, yet there is the least impediment of maternal venous return. These investigators determined that an angle of 27° on an inclined board provided the most effective position for chest compressions with the least impact on venous return. The current recommendation for positioning in pregnant patients during cardiac arrest is elevation of the right hip at 15 to 30° with a resuscitation wedge. Treating the mother takes precedence in this situation. The decision to perform postmortem caesarean section is a difficult one. Delivery of the infant could remove the compromising effects of aortocaval compression and restore venous return, while likewise removing a potentially viable fetus from an increasingly toxic environment. Many believe that a decision to perform a caesarean section should be made early, within the first 5 minutes, because the optimal infant and maternal survival occurs when the interval from arrest to delivery is <4 minutes.

#### **REFERENCES (8)**

American Heart Association. Cardiac arrest associated with pregnancy. *Circulation* 2005;112:IV-150. Mallampalli A, Guy E. Cardiac arrest in pregnancy and somatic support after brain death. *Crit Care Med.* 2005;33(suppl):S325-S331.

Rees GAD, Willis BA. Resuscitation in late pregnancy. Anesthesia. 1998; 43:347-349.

RATIONALE (9)

Answer: D

Amniotic fluid and venous air embolism may present with sudden cardiovascular collapse. Both may occur during labor. Amniotic fluid embolism is usually associated with a prolonged or difficult labor, and venous air embolism may be associated with a normal labor with placenta previa. Both types of embolism may result in release of inflammatory mediators and pulmonary vasoconstriction with diffuse pulmonary infiltrates. The presence of DIC is more typical of amniotic fluid embolism. If one survives the early cardiorespiratory collapse, DIC with frank bleeding commonly occurs over the next 1 to 2 hours.

#### **REFERENCES (9)**

Gei AF, Vadhera RB, Hankins GDV. Embolism during pregnancy: thrombus, air, and amniotic fluid. Anesthesiology Clin N Am. 2003;21:165-182.

Jiva TM. Critical care of pregnant women, part 2: fluid and air embolism, pneumonia. *J Crit Illness*. 2000;15:424-429.

Moore J, Baldisseri M. Amniotic fluid embolism. *Crit Care Med.* 2005;33(suppl):S279-S285. Tuffnell DJ. Amniotic fluid embolism. *Curr Opin Obstet Gynecol.* 2003;15:119-122.

RATIONALE (10)

Answer: A

This patient has developed a hepatic hematoma (exemplified by the less dense crescent shaped rim around the lateral aspect of the liver on the CT scan shown) as a complication from HELLP syndrome. HELLP syndrome is characterized by the following clinical findings: (1) elevated liver enzymes (AST and ALT); (2) microangiopathic hemolytic anemia; and (3) thrombocytopenia (platelet count <100,000/mm³). Patients develop HELLP syndrome in the third trimester of pregnancy, and up to 30% may develop the syndrome postpartum. Typical presenting symptoms are abdominal pain (RUQ), malaise, nausea, and vomiting. Complications include DIC, placental abruption, renal failure, and liver hematomas. Treatment for HELLP syndrome has traditionally involved expeditious termination of pregnancy. Conservative treatment may be indicated in selected cases allowing improved fetal maturity. Treatment of postpartum HELLP syndrome is mostly supportive. The most feared complications of this syndrome are liver subcapsular hematoma and rupture with shock. The CT scan shown in the question illustrates a hepatic hematoma with an intact capsule. Treatment for these cases is supportive. Transfusion of platelets (platelet count <50,000/mm³ with evidence of bleeding) and packed red blood cells are appropriate in this case. Surgery is reserved for cases of rupture and shock. There is no indication for a percutaneous drainage, which could lead to serious complications. With a normal partial thromboplastin time and international normalized ratio, fresh frozen plasma is not indicated. Steroids have been used in HELLP syndrome with mixed results, but a randomized, blinded trial showed no improvement in outcomes. Plasmapheresis has been recommended in HELLP syndrome lasting >72 hours postdelivery based on case reports. Although possibly helpful in some circumstances, the use of plasma exchange would not be the first treatment option in this case.

### REFERENCES (11)

Budev MM, Arroliga AC, Falcone T. Ovarian hyperstimulation syndrome. *Crit Care Med.* 2005;33(suppl):S301-S306.

Enskog A, Henriksson M, Unander M, et al. Prospective study of the clinical and laboratory parameters of patients in whom ovarian hyperstimulation syndrome developed during controlled ovarian hyperstimulation for in vitro fertilization. *Fertil Steril*. 1999;71: 808-814.

The Practice Committee of the American Society for Reproductive Medicine. Ovarian hyperstimulation syndrome. *Fertil Steril*. 2003;80:1309-1314.

RATIONALE (12)

Answer: D

There are a number of important physiologic (and specifically hemodynamic) effects of pregnancy of which the critical care clinician should be aware. These would especially be important if a pregnant patient would to become hemodynamically unstable and need cardiovascular support. Pregnancy is associated with an increase of total circulating blood volume (as much as 1,500 mL). It is notable that patients with pre-eclampsia may not have this typical degree of increased circulating blood volume and, therefore, may be at higher risks for hemodynamic effects with blood loss associated with delivery. The gravid uterus can decrease venous return to the heart when the patient is in the supine position. Therefore, for a pregnant patient with signs of acute circulatory insufficiency, it is important to place the patient in the left lateral decubitus position to facilitate venous return to the heart. Despite an increase in total circulating blood volume, there is no demonstrable increase in pulmonary capillary occlusion pressure. The HR is typically mildly elevated. Normal physiology of pregnancy will exhibit a decrease in systemic vascular resistance.

# **REFERENCE (12)**

Yeomans ER, Gilstrap LC, 3rd. Physiologic changes in pregnancy and their impact on critical care. Crit Care Med. 2005;33(10 suppl):S256-S258. RATIONALE (13)

Answer: D

The half-life of low-molecular-weight heparin (LMWH) is decreased in pregnancy, therefore twice-daily dosing is preferred rather than once daily dosing that can be used in nonpregnant individuals. The dose should be adjusted for weight or by measuring antifactor Xa levels. Unfractionated heparin and LMWH are equivalent in treatment of venous thrombosis, but LMWH is easier to administer and less likely to cause heparin-induced thrombocytopenia. Coumadin is only relatively contraindicated after the first trimester. Anticoagulation should be continued for approximately 6 weeks after delivery in the absence of other risk factors. An inferior vena cava filter is not routinely recommended because of potential worsening of venous thrombosis.

#### **REFERENCES (13)**

Bates SM, Greer IA, Hirsch J, Ginsberg JS. Use of antithrombotic agents during pregnancy. *Chest.* 2004; 126(suppl 3):627S-644S.

Gei AF, Vadhera RB, Hankins GDV. Embolism during pregnancy: thrombus, air, and amniotic fluid. *Anesthesiology Clin N Am.* 2003;21:165-182.

Toglia MR, Weg JG. Venous thromboembolism during pregnancy. N.Engl J Med. 1996; 335:108-114.

# SECTION 9: Neurologic Critical Care

# SECTION 9: NEUROLOGIC CRITICAL CARE

Instructions: For each question, select the most correct answer.

1. A 50-year-old male is brought to the emergency department by paramedics after collapsing at a local tennis club. A bystander had provided CPR, and on arrival to the scene the paramedics successfully cardioverted the patient from ventricular fibrillation. Patient is now unresponsive to stimuli and currently receiving mechanical ventilation with the following vital signs: temperature 37.4°C (99.3°F), HR 110/min, BP 135/78 mm Hg, and RR 20/min.

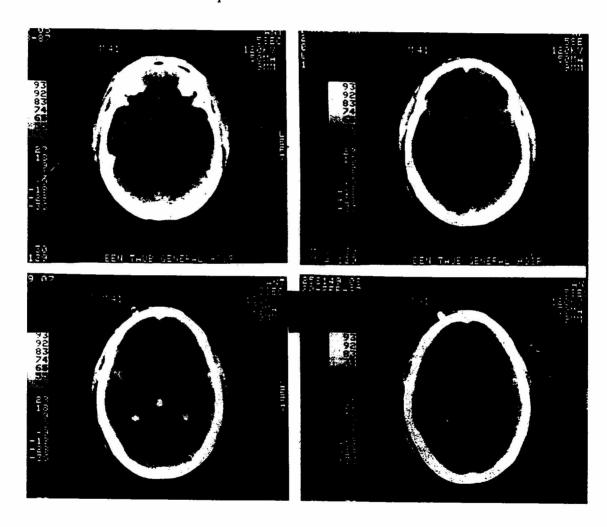
Which of the following interventions is most likely to improve neurologic outcome in this patient?

- A. Amiodarone (1 mg/min for 6 hours then 0.5 mg/min)
- B. Insulin to keep glucose 80 to 110 mg/dL
- C. Primary seizure prophylaxis with phenytoin
- D. Hypothermia (32 to 34 C°) for 12-24 hours
- E. β-Blockade

2. You are asked to evaluate a 52-year-old male who presented to the emergency department with depressed level of consciousness. On examination, the patient is lethargic, responds to painful stimuli, and is able to move all extremities. Vital signs are temperature 37.3 C° (99.1°F), HR 98/min, BP 220/105 mm Hg, and RR 20/min. A noncontrast CT scan of the head is shown below.

Which of the following is the most appropriate intervention for this patient?

- A. Decrease BP to 120/80 mm Hg with a titratable IV antihypertensive
- B. Initiate heparin infussion
- C. Decrease mean arterial pressure by 20% with a nitroprusside IV infusion
- D. Do not lower the BP at this point



3. You are asked to evaluate a 60-year-old male who presented to the emergency department with complaints of right lower and upper extremity weakness. The symptoms started 2 hours ago. Over this time period, the patient's wife reports that his weakness has gotten worse and that he has developed slurred speech. The patient has a history of hypertension and diabetes and was recently (2 weeks ago) hospitalized for a gastrointestinal hemorrhage.

Vital signs are temperature 37.1°C (98.8°F), HR 98/min, BP 187/98 mm Hg, and RR 14/min. On examination, the patient has slurred speech and is weak on the right side (upper extremity 1/5 and lower extremity 0/5). Laboratory data reveal normal electrolytes, normal complete blood count, and an international normalized ratio of 1.4. A CT scan of the brain without contrast was done and is shown below.

Which of the following interventions is most appropriate at this point?

- A. Lower BP to <150/90 mm Hg, and administer systemic thrombolytics (rT-pa)
- B. Lower BP to 120/80 mm Hg, and do not administer systemic thrombolytics (rT-pa)
- C. Do not treat BP, and administer systemic thrombolytics (rT-pa)
- D. Do not treat BP, and do not administer thrombolytics (rT-pa)
- E. Administer fresh frozen plasma, lower BP to <140/90 mm Hg, and administer systemic thrombolytics (rT-pa)



- 4. In a patient with an acute aneurysmal subarachnoid hemorrhage, which of the following findings on physical examination should alert the physician to associated increased intracranial pressure?
  - A. Meningismus
  - B. Third-nerve palsy
  - C. Sixth-nerve palsy
  - D. Bilateral lower-extremity weakness
  - E. Hemiparesis
- 5. A 65-year-old female admitted to the ICU after endovascular coiling of a ruptured posterior communicating artery aneurysm with subarachnoid hemorrhage develops new focal neurologic deficits and change in mental status on day 8 of her hospitalization.

Which of the following findings on admission is the best predictor for the occurrence of this complication?

- A. Amount of blood on CT scan
- B. Presence of focal neurologic deficits
- C. Localization of ruptured aneurysm
- D. Level of consciousness
- E. Elevated BP

6. A 40-year-old man is admitted to the ICU with inability to walk that progressed over the preceding 24 hours. Vital signs are BP 140/80 mm Hg, pulse 88/min, RR 20/min, and temperature 37.2°C (99.0°F). He is alert and oriented but complains of cramping pain in his legs bilaterally with paresthesias.

Neurologic examination shows 2/5+ strength in the proximal and distal muscle groups of the lower extremities and absent ankle and knee deep tendon reflexes. Rectal sphincter tone is normal. He has normal strength of his upper extremities and cranial nerves are normal.

Which one of the following interventions is the most appropriate initial therapy to improve neurologic outcome with the least adverse effects?

- A. Plasmapheresis and concomitant IV immunoglobulin
- B. Plasmapheresis followed by IV immunoglobulin
- C. IV immunoglobulin
- D. High-dose corticosteroids
- 7. A 37-year-old female with a history of myasthenia gravis presents after a week-long upper respiratory tract infection. She is noted to have a nasal voice, weak gag, and coughing with any swallow of saliva. She is intubated due to concerns for airway protection. She has a course of methylprednisolone initiated at 1 mg/kg. She begins to show improvement after the first day.

Assuming weaning criteria is met, which one of the following timings of extubation is the most appropriate, considering both the chance of successful extubation versus the risk of continued intubation?

- A. Two days after the initiation of methylprednisolone therapy
- B. One week after the initiation of methylprednisolone therapy and 1 hour after administration of pyridostigmine
- C. One week after the initiation of methylprednisolone therapy and 8 hours after administration of pyridostigmine
- D. When the patient is not receiving steroid therapy and pyridostigmine

8. A 75-year-old woman with a past medical history of breast cancer presents to an emergency department after being found unresponsive. She was last seen well 1 day prior by her daughter. On admission to the ICU, she is intubated and mechanically ventilated but not sedated. Her examination shows clear lung fields, a normal cardiac examination, and normal abdomen examination.

There is no evidence of pressure sores. Her cranial nerve examination shows normal oculocephalic reflexes. Her blood examination shows a normal creatinine kinase level, a normal arterial blood gas, normal chemistry panel, and a normal CT scan of the head. An MRI of the head is not available in the hospital.

Which one of the following is the most appropriate next step?

- A. A biopsy of the brain
- B. Comprehensive blood hepatic profile
- C. Bone scan
- D. Electroencephalogram
- E. Therapeutic trial with corticosteriods
- 9. A 65-year-old female with an unknown medical history is admitted to the hospital through the emergency department with complaints of fever, shortness of breath, productive cough, and myalgias. Chest radiograph on admission shows a right lower lobe infiltrate and IV antibiotics are started for the treatment of a community-acquired pneumonia. Laboratory studies showed an elevated white blood cell count with a neutrophil predominance, a mild transaminitis, hypoalbuminemia, and the presence of an anemia, and thrombocytopenia and an elevated prothrombin time/partial thromboplastin time. Over the night, the patient is noted to be confused, agitated, and paranoid. The nurses note that she is making up vivid and elaborate stories to account for her presence in the hospital. On examination, she was awake, alert with fluent speech, and moving all extremities equally; she does not cooperate with the cranial nerve examination but shows limitation of eye movements.

Which one of the following is the appropriate first step in this patient's care?

- A. Administer IV antibiotics (ceftriaxone/vancomycin/metronidazole), and perform a lumbar puncture
- B. Administer IV thiamine
- C. Obtain an urgent psychiatry consult
- D. Obtain a brain MRI and MR angiogram
- E. Administer an amp of IV dextrose solution

10. A male patient developed seizures and coma several days postoperatively after removal of a brain tumor and placement of a ventriculostomy for decompression of hydrocephalus. Laboratory values include sodium 112 mmol/L, potassium 4.1 mmol/L, chloride 80 mmol/L, HCO<sub>3</sub> 26 mmol/L, blood urea nitrogen 28 mg/dL, and creatinine 1.0 mg/dL. Ventilator settings are F1O<sub>2</sub> 0.6, PEEP 12 cm H<sub>2</sub>O, and pressure control mode at 35 cm H<sub>2</sub>O pressure. The patient lost 10% of body weight postoperatively. Urine output is 3 to 4 mL/kg/h, and urine sodium is measured at 170 mmol/L. A urinalysis is normal. Vital signs reveal HR 112/min, BP 110/70 mm Hg, RR 14 (on mechanical ventilation), and temperature 36.7°C (98.0°F).

Which one of the following is the most likely diagnosis?

- A. Diabetes insipidus
- B. Syndrome of inappropriate ADH (SIADH)
- C. Iatrogenic intoxication
- D. Cerebral salt wasting syndrome
- E. High output renal failure
- 11. A 25-year-old graduate student is transferred to the ICU from the Student Health Service after presenting with changes in mental status and a seizure. The patient had returned from a white water rafting vacation with friends when it was noted that he had onset of an acute febrile illness characterized by headache, disorientation, and behavioral abnormalities. He was aphasic and appeared to have hallucinations prior to presentation. He was noted to have right arm motor weakness and a focal seizure confined to that area. In the ICU, the patient progressed to unresponsiveness. There was no significant past medical history, and the patient did not have any travel history outside of the United States in the last 10 years. Vital signs revealed temperature 39.2°C (102.6°F) rectally, RR 18/min, HR 106/min, and BP 110/70 mm Hg. On physical examination, the patient is unresponsive with a slightly rigid neck. His pupils are midposition, reactive to light, and optic disks appeared to be flat. Heart, lung, and abdominal examinations are normal. The neurologic examination reveals hyperreflexia in the right upper extremity with an upgoing toe on that side. Cerebral spinal fluid from a lumbar puncture reveals a white blood cell count of 200 cells/mm³, predominantly monocytic. The protein is mildly elevated and the glucose is normal Gram stain is negative, and there are noted to be approximately 1,000 red blood cells/mm<sup>3</sup>.

Which one of the following is the most beneficial intervention for this patient?

- A. Dexamethasone followed by ceftriaxone and vancomycin
- B. Stat neurosurgical evaluation and cerebral angiogram
- C. Acyclovir
- D. Amphotericin B
- E. Hold antimicrobial agents and perform another lumbar puncture in 6 hours

12. A 64-year-old hypertensive patient presents with a spor	ntaneous intracerebral (intraparenchymal)
hemorrhage.	

Which one of the following locations of intracerebral hemorrhage would be most likely to require surgical intervention (ie, craniotomy)?

- A. Basal ganglia
- B. Cerebellum
- C. Internal capsule
- D. Parietal lobe (unilateral)
- 13. Which one of the following is not a risk factor for developing prolonged neuromuscular blockade following discontinuation of paralytic agent?
  - A. Depth of blockade
  - B. Coadministration of steroids
  - C. Hypomagnesemia
  - D. Renal failure
  - E. Male sex
- 14. Which one of the following is most correct concerning delirium?
  - A. Delirium typically shows a slow progressive and worsening course if left untreated
  - B. The presence of hallucinations makes delirium unlikely
  - C. The presence of a decreased level of motor activity makes delirium unlikely
  - D. Delirium is characterized by disturbances in the sleep cycle

- 15. Which one of the following statements is most correct concerning cardiac abnormalities occurring after subarachnoid hemorrhage?
  - A. ECG changes occur in less than one third of patients with subarachnoid hemorrhage
  - B. When creatine kinase MB isoenzymes and troponins are elevated, they approach levels typically seen in traditional ischemic myocardium
  - C. When echocardiographic wall motion abnormalities are present, they typically do not match vascular distribution
  - D. Decreased ejection fraction, occurring secondary to subarachnoid hemorrhage, is long lasting and often permanent if the patient survives
- 16. A 55-year-old male is admitted to the ICU after a closed head trauma. He has a depressed level of consciousness, with stable vital signs. He opens his eyes to speech. He localizes to pain. He moans but does not speak. There are no lateralizing signs.

What is his Glasgow coma scale score?

- A. 15
- B. 13
- C. 10
- D. 8
- E. 6
- 17. Which one of the following negates the use of the apnea test to confirm the presence of brain death?
  - A. Mean arterial pressure of 75 mm Hg on norepinephrine of 3 μg/min
  - B. PaCO<sub>2</sub> of 50 mm Hg
  - C. PaCO, of 30 mm Hg
  - D. Hypothermia

18. A 35-year-old male sustained a cardiac arrest secondary to stimulant abuse. Following 20 minutes of CPR, he regained a pulse. He is now intubated in the ICU on no medication and has been hemodynamically stable for the last 48 hours. His head CT scan reveals no focal lesion, and his HR is 110/min, with a mean arterial pressure of 75 mm Hg. On examination, you find no evidence of cortical or cranial nerve activity. When a painful stimulus is applied to the sole, the patient exhibits occasional foot movement.

In light of these foot movements, which one of the following is the most appropriate consideration at this time?

- A. Raise his mean arterial pressure to ≥85 mm Hg and follow-up with a neurologic examination
- B. Obtain a repeat CT scan of the head
- C. Initiate benzodiazepine therapy
- D. Foot movement due to painful stimulation does not preclude declaration of brain death
- 19. A 66-year-old male has a CT scan demonstrating a large intracerebral hemorrhage in the right thalamus. He has a hemiparesis of the left face, arm, and leg. His ECG shows inverted symmetric T waves over the precordial leads. Vital signs are: BP 170/100 mm Hg; HR 80/min; RR 18/min; afebrile. Laboratory studies reveal a normal complete blood cell count and metabolic panel, with glucose levels of 300 mg/dL and an international normalized ratio of 1.3.

Which one of the following is the most appropriate next step in this patient's care?

- A. Consult a cardiologist for possible coronary angiogram and place an IV nitroglycerin drip
- B. Monitor the BP and cardiac status, and reserve treatment or consultation for after ECG findings
- C. Administer labetalol to lower the BP to 140 systolic
- D. Administer an IV nitroprusside drip to lower the BP by 15-20%

20. Which one of the following volume pressure curves is most indicative of an increase in the total volume of intracranial contents?

Α.



В.



C.





D.

- 21. Which one of the following is not a potential confirmatory test for brain death?
- A. Arteriography
- B. Radioisotope study
- C. Magnetic resonance imaging
- D. Transcranial Doppler ultrasonography
- E. Electroencephalography

- 22. Which one of the following is most correct concerning a massive middle cerebral artery (MCA) infarction (involving ≥50% of the right or left cerebral hemisphere)?
  - A. Cerebral edema is predominately vasogenic
  - B. The primary cause of death is cerebral herniation
  - C. Potential benefit of hemicraniectomy is optimized when performed following failure of neurologic examination to improve by 48 hours
  - D. Cerebral edema related mortality is highest during the second week following infarction

# **SECTION 9: NEUROLOGIC CRITICAL CARE**

#### **ANSWERS:**

1-D; 2-A; 3-D; 4-C; 5-A; 6-C; 7-C; 8-D; 9-B; 10-D; 11-C; 12-B; 13-E; 14-D; 15-C; 16-C; 17-D; 18-D; 19-B; 20-D; 21-C; 22-B

RATIONALE (1)

Answer: D

The current advanced cardiac life support guidelines make an important emphasis on appropriate postresuscitation support, in an effort to improve outcomes of patients with cardiac arrest. Increasing attention has been placed on the institution of measures that may improve long-term, neurologically intact survival. Among the choices provided in the question, the correct answer is D, hypothermia (32-34°C) for 12-24 hours. The application of therapeutic hypothermia for patients with an out of a hospital cardiac arrest who have a return of spontaneous circulation and present to the hospital unresponsive have been studied in two, recently published, randomized, clinical trials. Both studies demonstrated that therapeutic hypothermia can improve long-term neurologic outcomes and survival in patients with ventricular fibrillation out-of-hospital cardiac arrest. Other important measures to improve outcomes include the aggressive treatment of seizures if present, and also control of hyperglycemia. Amiodarone and β-blockers have not been associated with improved neurologic outcomes in patients with cardiac arrest. Tight glycemic control has received a lot of attention in the last several years based on the findings of a study that targeted keeping the glycose between 80-110 mg/dL in a surgical critical care population and improved survival. No studies have demonstrated that tight glycemic control improved neurologic outcomes in patients postcardiac arrest. However, it is recognized that hypoglycemia can be deleterious to neurologic recovery, and it is recommended that it be treated. Although aggressive treatment of seizures when present is recommended by the advanced cardiac life support guidelines, there is no literature to support primary seizure prophylaxis in these patients.

### **REFERENCES (1)**

AHA ACLS Guidelines 2005. Part 7.5: postresuscitation support. Circulation 2005;112:84-85.

Hypothernia After Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med 2002;346:549-556.

Beranard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med* 2002;346:557-563.

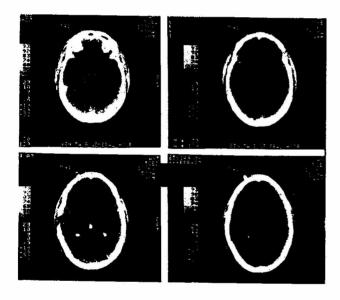
Polderman K. Application of therapeutic hypothermia in the intensive care unit. *Intensive Care Med* 2004;30:757-769.

Williams Gr, Spencer FC. The clinical use of hypothermia following cardiac arrest. *Ann Surg.* 1958;148:462-468.

RATIONALE (2)

Answer: A

The patient in this question has suffered a subarachnoid hemorrhage, most likely secondary to a ruptured aneurysm. The CT scan of the brain shown in the figure illustrates blood in the subarachnoid space (both in the basilar cisterns [small arrows] and in the sulci [large arrows]). All patients with subarachnoid should be evaluated and treated emergently. After initial stabilization, the main goals of treatment are the prevention of rebleeding, the prevention and management of vasospasms, and treatment of other medical complications associated with subarachnoid hemorrhages. Management of BP in patients with subarachnoid hemorrhage depends greatly on the status of the aneurysm. Before an intervention such as clipping or endovascular coiling is performed to secure the aneurysm, BP should be maintained within normal limits. To this effect, if necessary, IV antihypertensive agents, such as nitroprusside, labetalol, or nicardipine, labetalol should be used. Once the aneurysm is secured, hypertension is allowed. However, there is no agreement on the safest range of BP. In this patient with an unsecured aneurysmal bleed, the initial goal should be to normalize the BP, in order to decrease the incidence of rebleeding. Heparin is contraindicated with any intracranial acute hemorrhage. Answer C, decrease the mean arterial pressure by 20% with nitroprusside is appropriate to most hypertensive emergencies, however, in this specific case, the goal is to normalize the BP. It is also important to remember that calcium antagonist can reduce the risk of poor outcome from ischemic complications later on in the course of subarachnoid hemorrhages. Currently, oral nimodipine is recommended.



# **REFERENCES (2)**

Bambakidis NC, Selman Wr. Subarachnoid hemorrhage. In: Suarez JI, ed. Critical care neurology and neurosurgery. Totowa, NJ: Humana Press, 2004:365-377.

Brisman JL, Song JK, Newell DW. Cerebral aneurysms. N Engl J Med 2006;355:928-39.

Broderick J, Connolly S, Feldmann E, et al. Guidelines for the management of spontaneous intracerebral hemorrhage in adults. *Stroke* 2007;38:2001-2023.

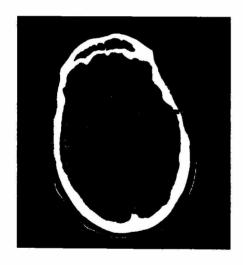
Suarez J, Tarr R, Selman W. Aneurysmal Subarachnoid Hemorrhage. *N Engl J Med* 2006;354:387-396. Suarez JI, Tarr RW, Selman WR. Aneurysmal subarachnoid hemorrhage. *N Engl J Med* 2006;354:387-96.

Torner JC, Kassell NF, Wallace RB, et al. Preoperative prognostic factors for rebleeding and survival in aneurysm patients receiving antifibrinolytic therapy: a report of the cooperative aneurysm study. *Neurosurgery* 1981;9:506-513.

RATIONALE (3)

Answer: D

The patient in this question is suffering an acute ischemic stroke. The CT scan shows an acute occlusive thrombus of the left MCA territory (see arrow), with no evidence of intracranial hemorrhage. Management of hypertension in the setting of an acute stroke remains difficult. On one hand, there are concerns of increased damage secondary ischemia and edema formation with uncontrolled hypertension. These concerns need to be balanced with potential hypoperfusion from aggressive lowering of BP in a situation where cerebral flow becomes dependant on perfusion pressures secondary to the loss of autoregulatory function in the cerebral vasculature. For ischemic stroke, current practice is to allow higher BPs to assure perfusion of viable cerebral tissue. The one consideration to be made is whether the patient is a candidate for thrombolytic therapy. In patients who will receive thrombolytics, BP should be lowered to systolic BP ≤180 mm Hg and a diastolic blood pressure ≤110 mm Hg. This patient is not a candidate for thrombolytic therapy secondary to a recent history of gastrointestinal hemorrhage. Therefore, management of his hypertension should be much more conservative and in accordance with current recommendations should be only treated if systolic BP is over 220 mm Hg or a diastolic blood pressure over 120 mm Hg. Because of the contraindication for thrombolytics, choices A, C, and E are incorrect. Choice B is incorrect, because, in the setting of an acute ischemic stroke, it is incorrect to lower the BP acutely to normal values because of potential ischemia to the permubra salvageable brain area surrounding the stroke.



# **REFERENCES (3)**

Adams HP, Adams RJ, Brott TG, Del Zoppo G, Furlan AJ, et al. Guidelines for the early management of patients with ischemic stroke: a scientific statement from the Stroke Council of the American Stroke Association. *Stroke* 2003;34:1056-1083.

Adams HP, Brott TG, Crowell RM, Furlan AJ, Gomez CR. Guidelines for the management of patients with acute ischemic stroke: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Circulation* 1994;90:1588-1601.

Adams HP, del Zoppo G, Alberts MJ, et al. Guidelines for the early management of adults with ischemic stroke. *Stroke* 2007;38:1655-1711.

Marik PE, Varon J. Hypertensive crises. Chest 2007;131:1949-1962.

Varon J, Marik P. Clinical review: The management of hypertensive crises. *Critical Care* 2003;7:374-384.

RATIONALE (4) Answer: C

The typical clinical presentation of subarachnoid hemorrhage includes the sudden onset of severe headache (frequently described as "the worse headache ever"), nausea, vomiting, neck pain and alterations of consciousness. Physical examination of patients presenting with subarachnoid hemorrhage may reveal retinal hemorrhages, signs of meningismus, diminished levels of consciousness, and localizing focal neurologic signs. Among the focal neurologic signs, some that are important mentioning include a third-nerve palsy that can be associated with a posterior communicating aneurysm, sixth-nerve palsy associated with increased intracranial pressure, bilateral lower-extremity weakness seen with anterior communicating aneurysms, and a combination of hemiparesis and aphagia or visual spatial neglect that might indicate a middle cerebral artery aneurysm. These findings can sometimes help localize possible sources of the subarachnoid hemorrhage or may indicate complications, such as increased intracranial pressure.

# **REFERENCE (4)**

Suarez J, Tarr R, Selman W. Aneurysmal subarachnoid hemorrhage. N Engl J Med 2006;354:387-396.

RATIONALE (5)

Answer: A

The patient in this question is showing clinical signs consistent with the development of symptomatic vasospasm as a complication of her subarachnoid hemorrhage. Symptomatic vasospasm is a common complication of subarachnoid hemorrhage (46% of patients) and usually develops between days 4 and 12 after the initial subarachnoid hemorrhage. It is believed that the arterial vasospasm is caused by an inflammatory reaction in the blood vessel wall. Angiographic vasospasm is more common than symptomatic vasospasm with clinical evidence of cerebral ischemia. Daily monitoring with transcranial doppler ultrasonography has been proposed to identify vasospasm in its early phases and intervene accordingly. Once symptomatic vasospasm develops (with evidence of focal neurologic signs, as in this patient), patients should be treated with hypervolemia and induced hypertension to maintain cerebral perfusion. Patients who fail medical therapy may require emergency cerbral angiography and transluminal angioplasty or vasodilator infusion to improve symptoms. Among the predictors on admission for the development of vasospasms the best one is the amount of blood seen on the initial head CT scan.

# **REFERENCES (5)**

Fisher CM, Kistler JP, Davis J. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computed tomographic scanning. *Neurosurgery* 1980;6:1-9.

Hijdra A, Van Gijn J, Nagelkerke NJ, Vermeulen M, Van Crevel H. Prediction of delayed cerebral ischemia, rebleeding, and outcome after aneurysmal subarachnoid hemorrhage. *Stroke* 1988;19:1250-1256.

Suarez J, Tarr R, Selman W. Aneurysmal subarachnoid hemorrhage. *N Engl J Med* 2006;354:387-396. Report of World Federation of Neurological Surgeons committee on a universal subarachnoid hemorrhage grading scale. *J Neurosurg* 1988;68:985-986.

RATIONALE (6) Answer: C

This patient most likely has Guillain-Barré syndrome as manifested by an ascending motor paralysis with loss of deep tendon reflexes. The treatment of choice is either plasmapheresis or IV immunoglobulin. The combination of the two treatments has not been found to improve outcome more than a single intervention and exposes the patient to increased risks. Greater adverse effects are associated with plasmapheresis, compared with IV immunoglobulin. Corticosteroids do not affect the neurologic outcome in Guillain-Barré syndrome.

#### **REFERENCES (6)**

Hughes RAC, Cornblath DR. Guillain-Barré syndrome. Lancet 2005;366:1653.

Hughes RAC, Rafael JC, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome (review). *Cochrane Database of Systematic Reviews* 2006, Issue 1, Art. No. CD002063.

Rafael JC, Chevret S, Hughes RAC, Annane D. Plasma exchange for Guillain-Barré syndrome (review). *Cochrane Database of Systematic Reviews* 2002, Issue 2, Art. No. CD0017989. van der Meché FGA, Schmitz PIM, et al. A randomized trial comparing intravenous immune globulin and plasma exchange in Guillain-Barré syndrome. *N Engl J Med* 1992;1123-1129.

RATIONALE (7)

Answer: C

Patients with myasthenic crisis are intubated for a combination of muscular respiratory failure and decreased muscle tone in the pharyngeal muscles causing a danger of aspiration and pneumonia. The decision to extubate a myasthenic patient is predicated on the patient's improvement in both respiratory muscle and pharyngeal strength but must also take into account the typical course of the disease and treatment. The treatment of myasthenic crisis includes supportive care for autonomic dysfunction and respiratory dysfunction. In addition, glucocorticosteroids have been shown to improve the long-term outcome of patients in crisis. It is important to consider that patients may experience a worsening of symptoms occurring between 4 to 6 days after initiation of steroid therapy.

Pyridostigmine is the drug of choice for symptomatic treatment of myasthenia gravis. It acts as a cholinesterase inhibitor but does not modulate the immune dysfunction responsible for the disease. In addition, in the critically ill population, the pharmacokinetics of the drug elimination is unpredictable and usually shorter than in the outpatient population. This is a very worrisome side effect, because the strengthening that occurs with the medicine may not last until the next dose. This may lead to extubation during the drug peak and subsequent weakening at the drug nadir (8 hours after administration). For this reason, patients should be extubated when they are medically able but also with consideration of the medications they are taking.

#### **REFERENCES (7)**

- Berrouschot J, Baumann I, Kalischewski P, Sterker M, Schneider D. Therapy of myasthenic crisis. Crit Care Med 1997; 25:1228-1235.
- Gracey DR, Divertie MB, Howard FM. Mechanical ventilation for respiratory failure in myasthenia gravis: two-year experience with 22 patients. *Mayo Clin Proc* 1983; 58:597-602.
- Johns TR: Long-term corticosteroid treatment of myasthenia gravis. *Ann NY Acad Sci* 1987; 505:568-583.
- Provencio JJ, Connors AF, Bleck TP. Update in nonpulmonary critical care: critical care neurology. Am J Resp Crit Care Med 2001; 164:341-345.
- Thomas CE, Mayer SA, Gungor Y, Swarup R, Webster EA, Chang I, Brannagan TH, Fink ME, Rowland LP. Myasthenic crisis: clinical features, mortality, complications, and risk factors for prolonged intubation. *Neurology* 1997; 48:1253-1260.

RATIONALE (8)

Answer: D

Patients who come to the emergency department in a coma can be categorized into a number of groups: metabolic disarray, overdoses and poisoning, vascular disease, infection, and seizures. Of these groups, a commonly overlooked is nonconvulsive status epilepticus. According to Towne and colleagues, 8% of patients evaluated for coma in their study were found to be in nonconvulsive status epilepticus. The evaluation of a patient with coma should include a search for metabolic abnormalities, drugs of abuse, an investigation of medications in the home, a search for infection including meningitis, and an evaluation by both physical examination and imaging for vascular disease. If all of these tests show no reason for the coma, nonconvulsive status epilepticus should be considered. The gold standard test is an electroencephalogram. Patients with previous stroke, brain tumors, and previous history of seizures are at particular risk of continuous seizures.

# **REFERENCES (8)**

Plum F, Posner JB. *The diagnosis of stupor and coma*. 3rd ed. Philadelphia, PA:Davis, 1980. Towne AR, Waterhouse EJ, Boggs JG, Garnett LK, Brown AJ, Smith JR Jr., DeLorenzo RJ. Prevalence of nonconvulsive status epilepticus in comatose patients. *Neurology* 2000; 54:340-345.

RATIONALE (9) Answer: B

The patient presents with a community-acquired pneumonia and is found to be confused, confabulating, and has no recent memories of her illness. Her examination shows limited eye movements, and she has evidence of malnutrition on her laboratory tests. With Korsakoff syndrome, patients typically present with anterograde memory impairment. Wernicke encephalopathy is characterized by confusion, gait ataxia, and oculomotor abnormalities in an alcoholic patient, but is now also appreciated to occur in any malnourished state including end-stage cancer, intractable vomiting after gastric reduction procedures, hyperemesis gravidarum, prolonged hospital courses on IV fluids, and in chronic hemodialysis patients. On MRI imaging, there is mammillary body shrinkage, and involvement of the medial thalamic and periaqueductal nuclei. Treatment consists of parenteral administration of thiamine 100 mg/day for several days, as the gastrointestinal mucosa is poor at absorption in the malnourished state. Glucose should not be administered before the thiamine, so as to avoid precipitating Wernicke or cause an early form of the disease to progress.

#### **REFERENCES (9)**

Ihara M, Ito T, Yanagihara C, Nishimura Y. Wernicke's encephalopathy associated with hemodialysis: Report of 2 cases and review of the literature. *Clin Neurol Neurosurg* 1999; 101:118-121.

Kumar PD, Nartsupha C, West BC. Unilateral internuclear ophthalmoplegia and recovery with thiamine in Wernicke syndrome. *Am J Med Sci* 2000; 320:278-280.

Ogershok PR, Rahman A, Nestor S, Brick J. Wernicke encephalopathy in nonalcoholic patients. *Am J Med Sci* 2002; 323:107-111.

Zubaran C, Fernandes JG, Rodnight R. Wernicke-Korsakoff syndrome. Postgrad Med J 1997; 73:27-31.

RATIONALE (10)

Answer: D

Cerebral salt wasting syndrome was first described in patients who had subarachnoid hemorrhage. It has been described in patients with brain tumors, carcinomatous meningitis, and head trauma. Patients become hyponatremic, lose weight, and develop hypovolemia. As volume status is an important distinguishing feature from SIADH, assessment of intravascular volume may be necessary to make the diagnosis. Renal and endocrine causes of sodium and water loss must also be excluded. In this patient, he became hyponatremic with output of large volumes of urine. Urine sodium was high, and his vital signs and laboratory are consistent with hypovolemia and prerenal azotemia. Hyperglycemia must be ruled out as a cause of low measured serum sodium. Treatment of cerebral salt wasting syndrome is focused on replacement of salt and volume with saline solutions, colloid, or both. Hypertonic salt solutions may be necessary in the early resuscitation phase to elevate the serum sodium level to higher levels. Fludrocortisone may be helpful in patients who continue to lose large amounts of sodium in their urine. Recovery usually occurs spontaneously over 3 to 4 weeks.

The most common cause of hyponatremia in patients with intracranial pathology is SIADH. Patients are generally normovolemic or hypervolemic with low sodium and normal to decreased blood urea nitrogen. Urine volumes are low, with an inappropriately high osmolarity in the face of low serum osmolarity. Some postoperative medications, such opiates, can contribution to SIADH by causing release of excessive amounts of antidiuretic hormone. Aggressive hydration with hypotonic fluids in the postoperative period can lead to water intoxication and significant hyponatremia. Urine volumes are normal to high, but urine osmolarity is appropriately low, with respect to low serum osmolarity. Diabetes insipidus is caused by lack of antidiuretic hormone or antidiuretic hormone effect on the kidney. It is characterized by severe renal water loss and hypernatremia. High output renal failure secondary to ischemia or nephrotoxic agents is characterized by losses of large volumes of urine with high sodium content and can lead to hypovolemia, dehydration, and hyponatremia. Urine is isosthenuric, blood urea nitrogen, and creatinine are elevated, and urinalysis reveals active sediment.

#### **REFERENCES (10)**

Bleck TP. Metabolic encephalopathy. In: Emergent and Urgent Neurology. Weiner WJ, Schulman LM, eds. Philadelphia, PA: Lippincott, 1999, 223-53.

Harrigan MR. Cerebral salt wasting syndrome. Crit Care Clin 2001; 17:125-138.

Maesaka JK, Gupta S, Fishbane S. Cerebral salt-wasting syndrome: Does it exist? *Nephron* 1999; 82:100-109.

RATIONALE (11)

Answer: C

This patient most likely has herpes simplex encephalitis based on the physical examination findings, the clinical presentation, and cerebrospinal fluid analysis. Viral encephalitis begins with the acute onset of a febrile illness. Additional findings include fever, headache, disorientation, alterations in behavior, and speech, as well as neurologic findings that occasionally may be focal. These clinical findings distinguish a patient with encephalitis from a patient with a viral meningitis picture. Characteristically, patients with viral meningitis have nuchal rigidity, headache, and photophobia but lack focal neurologic findings or significant changes in mental status. The lumbar puncture is characteristic revealing a cerebrospinal fluid pleocytosis with monocytic predominance, a normal glucose, and red blood cells suggesting herpes encephalitis. Herpes simplex virus has a predeliction for the temporal lobe and an EEG may reveal focal findings. In addition, CT scan or MRI may reveal changes in the temporal lobe area. For bacterial meningitis, Gram stain might be expected to have positive results. Although fungal meningitis would be a concern, this usually presents in a subacute or chronic manner.

Viruses characteristically gain access to central nervous system through either hematogenous or neuronal spread. It is felt that herpes simplex gains access to the central nervous system via a neuronal pathway with some suggestion that the olfactory tract may be the choice of access. Herpes simplex is the most common cause of nonepidemic acute focal encephalitis in the United States. Infections with this virus may occur any time of the year and is more common in patients <20 and >50 years old. Blood in the cerebrospinal fluid is characteristic of herpes simplex encephalitis. A definitive diagnosis is established by brain biopsy, although this is usually reserved for patients who fail therapy with acyclovir. Successful therapy depends upon early institution of acyclovir, based upon a high index of suspicion, along with clinical findings and cerebrospinal fluid values.

## **REFERENCES (11)**

Jackson AC. Acute viral infections. Curr Opin Neurol 1995; 8:170.

Lipton JD, Schafermeuer RW. Central nervous system infections: the usual and the unusual. *Emerg Med Clinics North Am* 1995;13:417-443.

Schmutzhard E: Viral infections of the CNS with special emphasis on herpes simplex infections. *J Neurol* 2001; 248:469-477.

RATIONALE (12)

Answer: B

Although spontaneous intracerebral (intraparenchymal) hemorrhage (ICH) is more than twice as common as spontaneous subarachnoid hemorrhage, there have been comparatively few randomized trials of ICH treatment. Furthermore, as treatment strategies worldwide have been widely divergent, there has been no consensus on medical or surgical management of ICH. In 1999, a writing group from the Stroke Council of the American Heart Association (AHA) reviewed the available data and published guidelines for ICH management.

The most common cause of ICH remains hypertension. Hypertensive hemorrhages secondary to small-vessel disease most often occur in the putamen, global pallidum, thalamus, internal capsule, deep periventricular white matter, pons, and cerebellum. Other causes of ICH include illicit drug abuse (usually cocaine), vascular malformations or aneurysms, hemorrhage into cerebral infarcts or brain tumors, or complications of thrombolytic or anticoagulant therapy.

Recommendations from the AHA Scientific Statement included the following:

- 1. The initial study of choice for suspected ICH is a noncontrast CT scan of the head.
- 2. Angiography should be considered for surgical candidates without a clear cause of hemorrhage (ie, young normotensive patients). However, angiography is not necessary for older hypertensive patients who have a deep hemorrhage in the basal ganglia, thalamus, cerebellum, or brain stem, if the CT scan does not suggest a structural lesion. MRI and MRA may be useful in diagnosing cavernous malformations and may obviate the need for angiography in selected patients.
- 3. Compared with management of ischemic stroke, hypertension in ICH should be treated more aggressively (without inducing hypotension), in order to reduce ongoing bleeding from small vessels.
- 4. Clinical deterioration is the most important indication for surgical intervention. Patients with a moderate or large lobar hemorrhage who are clinically deteriorating should be considered for craniotomy. Surgical management should also be considered for patients with relatively small cerebellar hemorrhages (~3 cm) who are clinically deteriorating or have brain stem compression and/or obstructive hydrocephalus. Nonsurgical management is appropriate for patients with small hemorrhages, mild neurologic deficits, very poor prognosis, or Glasgow coma scale score ≤4.

A cerebellar hemorrhage is more likely to require surgical intervention (ie, craniotomy), because there is less room in the posterior fossa for expansion of blood and brain edema. Brain stem compression could occur with relatively small hemorrhages.

### **REFERENCE (12)**

Broderick JP, Adams HP, Barsan WB, et al. Guidelines for the management of spontaneous intracerebral hemorrhage: a statement for healthcare professionals from a special writing group of the stroke council. *Stroke* 1999; 30:905-915.

RATIONALE (13)

Answer: E

Neuromuscular blockade has seen a marked decrease in its utilization over the last 5 to 10 years. This is primarily because of the concern of persistent neuromuscular blockade following discontinuation of paralytic agent. This is more likely to occur in patients who are also receiving therapeutic steroids and in women. This is also true in the presence of renal failure, because many as the paralytic agents either are dependent directly on renal clearance or have active metabolites that are dependent upon renal clearance. Hypomagnesemia and hypermagnesemia are risk factors for prolonged neuromuscular blockade. The depth of blockade is also very important and, as the depth the blockade increases, the risk for prolonged neuromuscular blockade increases. This is why it is important to monitor all patients who are receiving continuous infusion of paralytic agents with a peripheral nerve stimulator. Female (not male) sex is a risk factor; this is perhaps due to the same dosage given, even with the average female's smaller size. The twitch monitor applies an electrical stimulus either to the ulnar nerve or the temporal nerve, and 4 successive electrical stimuli are delivered. The number of muscle twitches indicates the depth of the blockade. The lightest blockade is 4 twitches, and the

heaviest is no twitches. It is recommended that the patients on continuous infusion of paralytic agents be maintained at a 2-twitch or greater state. If the neuromuscular blocking agent is being used for ventilator rate control (no assisted breathing), as is often the case (Severe Acute Respiratory Distress Syndrome with inverse ratio ventilation or severe asthma with severe auto-PEEP), eliminating assisted ventilation should be the primary target, and achieving this with 4 twitches is ideal.

#### **REFERENCES (13)**

Hund E. Myopathy in critically ill patients. *Crit Care Med* 1999; 27:2544-2547. Viby-Mogensen J. Why, how, and when to monitor neuromuscular function. *Minerva Anestesiologica* 1999; 65:239-244.

RATIONALE (14)

Answer: D

In direct contrast to dementia, which is a chronic confusional state, delirium is an acute confusional state. The diagnosis of delirium is primarily clinical, and is based on careful beside observation. Delirium is often unrecognized by physicians and nurses due to its fluctuating nature, its overlap with dementia, the lack of formal cognitive assessment, an under appreciation of its clinical consequences, and a failure to consider the diagnosis important. It occurs abruptly, usually over a period of hours or days, and is characterized by a fluctuating course with symptoms coming and going over a 24-hour period. There are also characteristic lucid intervals, as well as inattentiveness, with some difficulty in focusing. Disorganized thinking is usually present as well.

Illusions or hallucinations are seen in approximately one third of the patients. Psychomotor disturbances may be hyperactive or hypoactive, the latter characterized by a marked decrease in the level of motor activity. Sleep cycle disturbances are characteristic, with typical daytime drowsiness, nighttime insomnia, fragmented sleep and complete sleep cycle reversal. Emotional disturbances are common.

#### **REFERENCES (14)**

Cole MG. Delirium in elderly patients. Am J Geriatr Psychiatry 2004;12:7-21.

Inouye SK. Delirium in older persons. N Engl J Med 2006;354:1157-1165.

Inouye SK, Rushing JT, Foreman MD, Palmer RM, Pompei P. Does delirium contribute to poor hospital outcomes? A three-site epidemiologic study. *J Gen Intern Med* 1998;13:234-242.

Inouye SK, Schlesinger MJ, Lydon TJ: Delirium: a symptom of how hospital care is failing older persons and a window to improve quality of hospital care. Am J Med 1999;106:565-573.

Levkoff SE, Evans DA, Litpzin B, et al. Delirium: the occurrence and persistence of symptoms among elderly hospitalized patients. *Arch Intern Med* 1992;152:334-340.

Murray AM, Levkoff SE, Wetle TT, et al. Acute delirium and functional decline in the hospitalized elderly patient. *J Gerontol* 1993;48:M181-M186.

O'Keeffe S, Lavan J. The prognostic significance of delirium in older hospital patients. *J Am Geriatr Soc* 1997;45:174-180.

RATIONALE (15)

Answer: C

Cardiac abnormalities after a subarachnoid hemorrhage are common, and include ECG changes in 25-100% of patients; cardiac enzyme elevations in 17-37%; and left ventricular dysfunction by echocardiography in 8-30%. These abnormalities are typically alluded to as "cardiac stun" and result from excessive catecholamine release in response to intracranial hemorrhage.

Wall motion abnormalities of the ventricular wall that do not match the electrocardiographic vascular distribution of ischemia are typical. Levels of creatine kinase MB enzymes generally are only 1/10 of that seen in true ischemic myocardium. Most cases of cardiac stunning are temporary, with permanent dysfunction rare, irrespective of whether early treatment of the aneurysm is performed.

### **REFERENCES (15)**

- Brisman JL, Song JK, Newell DW. Cerebral Aneurysms. N Engl J Med 2006; 355:928-939.
- Bulsara KR, McGirt MJ, Liao L, et al. Use of the peak troponin value to differentiate myocardial infarction from reversible neurogenic left ventricular dysfunction associated with aneurysmal subarachnoid hemorrhage. *J Neurosurg* 2003;98:524-528.
- Deibert E, Barzilai B, Braverman AC, et al. Clinical significance of elevated troponin I levels in patients with nontraumatic subarachnoid hemorrhage. *J Neurosurg* 2003;98:741-746.
- Jain R, Deveikis J, Thompson BG. Management of patients with stunned myocardium associated with subarachnoid hemorrhage. Am J Neuroradiol 2004;25:126-129.
- Khush K, Kopelnik A, Tung P, et al. Age and aneurysm position predict patterns of left ventricular dysfunction after subarachnoid hemorrhage. J Am Soc Echocardiogr 2005;18:168-174.
- Tung P, Kopelnik A, Banki N, et al. Predictors of neurocardiogenic injury after subarachnoid hemorrhage. *Stroke* 2004;35:548-551.
- Zaroff JG, Rordorf GA, Ogilvy CS, Picard MH. Regional patterns of left ventricular systolic dysfunction after subarachnoid hemorrhage: evidence for neurally mediated cardiac injury. *J Am Soc Echocardiogr* 2000;13:774-779.
- Zaroff JG, Rordorf GA, Newell JB, Ogilvy CS, Levinson JR. Cardiac outcome in patients with subarachnoid hemorrhage and electrocardiographic abnormalities. *Neurosurgery* 1999;44:34-39.

RATIONALE (16) Answer: €

The Glasgow coma scale (GCS) score was developed to provide a consistent description of patients with head injury, as well as provide a measure of severity. Patients are scored on eye opening, best motor response, and verbal response. Points are given for each area and the sum of the points provides the score. The best possible score is 15 and the worst score is 3.

The severity of head injury can be characterized as mild if it scores 14-15 on the GCS; moderate if the score is 9-13; and severe if the score is 3-8. Repeated examination and GCS scoring in response to treatment provides information for prognosis, morbidity, and mortality. A GCS score of 8 or less describes a coma; at that point, early tracheotomy is considered. The GCS is not a substitute for a complete neurologic examination with cranial and peripheral nerve evaluation for isolated or lateralizing signs.

By adding the scores highlighted on the scale below, it is clear that the patient described in this question has a score of 10, which indicates a moderate head injury.

#### Glasgow Coma Scale

Eye Opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Best Motor Response	Obeys commands	6
	Localizes pain	5
•	Withdraws normal flexion	4
	Decorticate abnormal flexion	3
	Decerebrate extension	2
	Flaccid no response	1
Verbal response	Oriented	5

#### **REFERENCES (16)**

Gennarelli TA, Champion HR, et al. Comparison of mortality, morbidity, and severity of 59,713 head-injured patients with 114,447 patients with extracranial injuries. *J Trauma* 1994;37:962-968. Head Trauma. ATLS. In ACS, 1997; 181-213.

Jennett H, Bond M. Assessment of outcome after severe brain damage: A practical scale. *Lancet* 1975;1:480-484.

RATIONALE (17)

Answer: D

The diagnosis of brain death is based on clinical neurologic criteria. In order to diagnose brain death the physician should establish: (1) the presence of an irreversible coma, (2) lack of brain stem reflexes, and (3) the presence of apnea. The first 2 are achieved by physical examination; the presence of apnea is established through an apnea test. In the United States, the threshold of maximal stimulation of the respiratory centers in the brain stem have been arbitrarily set at a partial arterial pressure of  $CO_2$  of 60 mm Hg or a value that is 20 mm Hg higher than the patient's baseline. A lack of spontaneous breathing after reaching these values constitutes a positive apnea test consistent with brain death.

In order to perform the apnea test, conditions that could confound the diagnosis of brain death must be excluded. These include hypothermia, drug intoxication/poisoning, and severe electrolyte imbalance. The patient must also be hemodynamically stable. Development of hemodynamic instability during the apnea test is an indication to abort the test and reconnect the patient to the ventilator.

Regardless of oxygen saturation, patients should be pre-oxygenated for apnea testing and receive 100% oxygen during the test. Apneic diffusion oxygenation is utilized to maintain oxygenation during the apnea test. Preoxygenation eliminates the stores of respiratory nitrogen and optimizes the transport of oxygen during the apnea test. This procedure is required prior to apnea testing since the occurrence of hypotension or arrhythmias during apnea tests are commonly related to hypoxemia and lack of oxygen during test or proper preoxygenation.

Body movements during testing can occur and are originated as spinal reflexes. Consistent clinical evidence of brain death should prevail in these cases. In the United States, the use of confirmatory tests such as an EEG or cerebral angiogram are not required if the above criteria are met in adults. However, in cases were there is doubt on the diagnosis, they may aid in establishing a definitive diagnosis of brain death.

### **REFERENCES (17)**

Gourdreau JL, Wijdicks EF, Emery SF. Complications during apnea testing in the determination of brain death: predisposing factors. *Neurology* 2000;55:1045-1048.

The Quality Standards Subcommittee of the American Academy of Neurology. Practice parameters for determining brain death in adults. *Neurology* 1995;45:1012-1014.

Wijdicks E. The Diagnosis of Brain Death. N Engl J Med 2001;344:1215-1221.

RATIONALE (18)

Answer: D

Approximately 30% of brain dead patients retain normal plantar response. Other spinal reflexes may also be present. Brain death (death by neurologic criteria) is defined as the complete and irreversible loss of both cortical and brain stem activities due to a known etiology. Reversible causes of brain dysfunction preclude the determination of brain death. These conditions may include drug intoxication (including sedatives such as benzodiazepine), hypothermia <89.6°F, hepatic encephalopathy, severe electrolyte or acid-base disorder, uremia, hypoglycemia, hypotension (mean arterial pressure <60 mm Hg), etc.

The presence of cocaine metabolites on a urine toxicology screen would not explain this patient's neurologic examination. However, benzodiazepines could potentially suppress cortical and brain stem activity. The cardiac index is of little significance for the determination of brain death as long as the blood pressure is adequately maintained. Declaration of brain death is based primarily on bedside clinical examinations. The patient should be unresponsive to painful stimuli (coma) and be without brain stem reflexes (including no oculovestibular reflexes to ice water calorics and no respiratory efforts during an adequate apnea test). A confirmatory test such as cerebral flow study, electroencephalogram, or cerebral angiogram is usually not necessary unless clinical findings are obscured by complicating factors. Specific criteria and regulations for declaration of brain death vary by state and institution.

#### **REFERENCES (18)**

Guidelines for the determination of death: report of the medical consultants on the diagnosis of death to the President's Commission for the study of ethical problems in medicine and biomedical and behavioral research. *JAMA* 1981;246:2184-2186

Kelly BJ, Luce JM. Neurologic criteria for death in adults. In Parrillo JE, Dellinger RP, eds. *Critical Care Med.* St. Louis, MO: Mosby; 2001:1278-1290.

Paolin A, Manuali A, DiPao LA, et al. Reliability and diagnosis of brain death. *Intensive Care Med* 1995;21:657-662.

Wijdicks EFM. Determining brain death in adults. Neurology 1995;45:1003-1011.

RATIONALE (19)

Answer: B

The patient appears to have suffered a hypertensive-related intracerebral hemorrhage, with a thalamic location being a common site. Given his age and no prior history of hemorrhages, a hemorrhage related to an underlying arteriovenous malformation is unlikely. The inverted T-waves on the ECG are likely cerebral in origin, as the T-waves are diffuse and symmetric and not accompanied by short-term segment elevation or depression.

Hypertension is common after an ischemic or hemorrhagic stroke in the first few days. The management of hypertension in the acute setting of an infarct is controversial, as there are no randomized, controlled clinical trials. With an intracerebral hemorrhage, there is a theoretical concern that high BP may enlarge the hemorrhage. There also exists some concern that lowering the BP may worsen the ischemia in the tissue surrounding the hemorrhage.

In chronic hypertensive patients, the cerebral autoregulatory curve is shifted rightward, such that acutely lowering the BP may decrease the cerebral perfusion pressure and bring about ischemia. There is no evidence that lowering BP in an intracerebral hemorrhage is beneficial. However, a recent study using positron emission tomographic imaging before and after BP modification did not reveal a significant change in the cerebral blood flow around the hematoma. This study, however, did not assess functional outcomes in the patients. Until more data are available from randomized clinical trials, however, for very high blood pressures (systolic values greater than 180-190), it is acceptable to either gently lower the mean arterial pressure by no more than 20% or allow the elevated blood pressure to remain unchanged.

### **REFERENCES (19)**

Fischberg GM, Lozano E, Rajamani K, Ameriso S, Fisher MJ. Stroke precipitated by moderate blood pressure reduction. *J Emerg Med* 2000;19:339-346.

Morgenstern LB, Yonas H. Lowering blood pressure in acute intracerebral hemorrhage: Safe, but will it help? *Neurology* 2001;57:5-6.

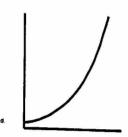
Powers WJ, Zazulia AR, Videen TO, et al. Autoregulation of cerebral blood flow surrounding acute (6 to 22 hours) intracerebral hemorrhage. *Neurology* 2001;57:18-24.

Qureshi AI, Tuhrim S, Broderick JP, Batjer HH, Hondo H, Hanley DF. Spontaneous intracerebral hemorrhage. N Engl J Med 2001;344:1450-1460.

RATIONALE (20)

Answer: D

The Monro-Kellie doctrine states that the combined total intracranial content volume (brain, blood, and cerebrospinal fluid [CSF]) remains constant as they are contained within the skull (a rigid compartment). As any one of these volumes increases, compensation occurs by the displacement of other parts of the intracranial components. Cerebral veins are compressed, resulting in decreased cerebral blood volume. The CSF volume also decreases, due to a combination of the displacement of CSF toward the spinal compartment and increased reabsorption. As compensatory mechanisms are exhausted and volume continues to increase, a sharp rise in intracranial pressure occurs, as demonstrated in the volume pressure curve in Figure D.



#### **REFERENCES (20)**

Go KG. Cerebral pathophysiology. Amsterdam, the Netherlands, Elsevier, 1991. Stocchetti N, Maas A, Chieregata A, van der Plas A. Hyperventilation in head injury. *Chest* 2005;127:1812-1827.

The Brain Trauma Foundation. The American Association of Neurological Surgeons: the Joint Section on Neurotrauma and Critical Care; Guidelines for cerebral perfusion pressure. *J Neurotrauma* 2000;17:507-511.

RATIONALE (21)

Answer: C

All published brain death criteria share four common elements: (1) irreversibility, (2) absence of neurologic function, (3) apnea, and (4) confirmation of the inability to adequately test brain stem reflexes (although this is controversial in some circumstances) or when the cause of brain death is not fully established or when it is desirable to shorten the period of observation. Potentially useful confirmatory tests include the following:

- (a) arteriography or radioisotope studies demonstrating no blood flow to the cerebrum, cerebellum, and brain stem;
- (b) transcranial Doppler ultrasonography showing no blood flow to the brain; and
- (c) electroencephalography EEG (without medications being administered that would produce isoelectric EEG).

Magnetic resonance imaging, although offering a precise view of brain anatomy, offers no useful information for presence or absence of brain death.

### **REFERENCES (21)**

- Ducrocq X, Braun M, Debouverie M, et al Brain death and transcranial Doppler: experience in 130 cases of brain dead patients, *J Neurol Sci* 1998;160: 41-46.
- George MS Establishing brain death: the potential role of nuclear medicine in the search for a reliable confirmatory test. *Eur J Nucl Med* 1999; 18:75-77.
- Kelly BJ, Luce JM. Neurologic criterial for death in adults. *In*: Parrillo JE, Dellinger RP, eds. Critical Care Medicine: Principles of Diagnosis and Management in the Adult. St. Louis, Mosby, 2001, pp 1278-1290.
- Report of the Quality Standards Subcommittee of the American Academy of Neurology. Practice parameters for determining brain death in adults (summary statement). *Neurology* 1995:45: 1012-1014.
- Wijdicks EFM. Determining brain death in adults. Neurology 1995;45:1003-1111.

RATIONALE (22)

Answer: B

Cerebral edema following massive cerebral infarction (MCA) is predominately cytotoxic and does not respond adequately to traditional medical therapy. In the great majority of patients, the cause of death is cerebral herniation and brain death. This deterioration is attributable to the mass effect and cerebral edema. Transtentorial herniation is representative of maximal cerebral edema. Hemicraniectomy has been proposed as a treatment modality to reduce the mass effect associated with MCA-induced cerebral edema. For maximum benefit, this intervention should be performed prior to significant secondary injury related to the cerebral edema. Hemicraniectomy performed within the first 24 hours appears to optimize potential benefit. The highest mortality is seen 3 to 6 days after a MCA infarction.

# **REFERENCES (22)**

- Delashaw JB, Broaddus WC, Kassell NF, et al. Treatment of right hemispheric cerebral infarction by hemicraniectomy. *Stroke* 1990; 21:874-881.
- Frank JI. Large Hemispheric infarction, deterioration, and intracranial pressure. *Neurology* 1995; 45:1286-1290.
- Hacke W, Schwab S, Horn M, et al. Malignant middle cerebral territory infarction: clinical course and prognostic signs. *Arch Neurol* 1996; 53:309-315.
- Qureshi, A.I. Timing of neurologic deterioration in massive middle cerebral artery infarction: a multicenter review. *Critical Care Medicine* 2003;31:272-277.

# SECTION 10: Pharmacology

# **SECTION 10: PHARMACOLOGY**

Instructions: For each question, select the most correct answer.

1. A 70-year-old male has been admitted to the ICU after a colectomy because of severe chronic obstructive pulmonary disease (COPD) and pain management. The procedure was uncomplicated, and the patient was extubated at the end of the procedure. On examination, the patient's RR is 16/min, and he rates his pain on a 0-10 scale as an 8.

Arterial blood gas values are as follows: pH 7.36, Paco<sub>2</sub> 45 mm Hg, PaO<sub>2</sub> 75 mm Hg, and HCO<sub>3</sub> 28 mEq/L while receiving oxygen at 4 L/min by nasal cannula.

Which one of the following analgesics for postoperative pain management would you select to minimize respiratory depression?

- A. Morphine
- B. Meperidine
- C. Butorphanol
- D. Hydromorphone
- E. Oxycodone
- 2. Which one of the following is *not* an expected adverse effect causally related to propofol use in adults for ICU sedation?
  - A. Myoclonic activity
  - B. Green urine
  - C. Hypotension
  - D. Supraventricular tachycardia
  - E. Hyperlipemia

- 3. Which one of the following is most correct concerning contraindications to use of specific intravenous antihypertensives in hypertensive emergencies?
  - A. Labetalol is contraindicated in first-degree heart block
  - B. Labetalol is contraindicated in diastolic left ventricular dysfunction
  - C. Hydralazine is contraindicated in dissecting aortic aneurysm
  - D. Phentolamine is contraindicated in pheochromocytoma
- 4. A 45-year-old female with chronic renal failure is admitted to the ICU with a hypertensive emergency. After 30 hours of treatment with nitroprusside, the patient develops confusion and metabolic acidosis. Administration of which of the following is most likely to prevent/treat these symptoms?
  - A. Thiosulfate
  - B. Cyanocobalamin
  - C. Pyridoxine
  - D. Glucogon
  - E. Calcium chloride
- 5. Which of the following statements is most correct in reference to the use of vasopressin in patients with septic shock?
  - A. Vasopressin will increase cardiac output and cardiac index
  - B. Vasopressin use in septic shock is associated with an increase in the Pco<sub>2</sub> gap between the gastric mucosa and arterial blood
  - C. Vasopressin use improves mortality in patients with septic shock
  - D. Vasopressin decreases platelet aggregation

- 6. Which of the following best describes the clinical antibacterial spectrum of tigecycline?
  - A. Gram-positive bacteria
  - B. Gram-positive bacteria + Gram-negative bacteria
  - C. Gram-negative bacteria
  - D. Gram-positive bacteria + Gram-negative bacteria + anaerobes
  - E. Gram-negative bacteria + anaerobic bacteria
- 7. You are taking care of a 56-year-old female with severe sepsis. The patient is being treated with antibiotics, sedatives, continuous insulin, and mechanical ventilation and is receiving recombinant human-activated protein C (rh-APC). The laboratory notifies you of a positive blood culture results for Gram-positive cocci. On examination, you notice that the insertion site of the right subclavian central venous catheter is erythematous and has a slight purulent discharge when pressed. Current laboratory data include white blood cell count 16,000, Hb 11.2, platelets 80,000 x 10<sup>3</sup>/μL, international normalized ratio 1.2, and activated partial thromboplastin time 30 seconds. You decide to change the central line.

Which of the following is the most appropriate approach?

- A. Hold rh-APC for 1 hour. Administer 2 U of fresh frozen plasma, and change line over a guide wire. Restart rh-APC 1 hour after an uncomplicated procedure
- B. Administer 1 U of platelets, 2 U of fresh frozen plasma, and place a new central catheter in the left subclavian vein
- C. Hold rh-APC for 2 hours, and place new central catheter in the left internal jugular vein. Restart rh-APC 2 hours after an uncomplicated procedure
- D. Hold rh-APC for at least 6 hours, transfuse platelets, and place a new line in the femoral vein. Do not restart rh-APC after an uncomplicated procedure

- 8. A 44-year-old male with HIV is admitted to the ICU with severe community-acquired pneumonia requiring mechanical ventilation. The patient is on an outpatient retroviral regimen that includes a protease inhibitor, lopinavir + ritonavir, and a nucleoside reverse-transcriptase inhibitor, zidovudine. The patient has done very well with this regimen (viral load, nondetectable), and his infectious disease physician recommends that he be kept on this regimen during his ICU hospitalization. Which of the following drugs should be avoided in this case?
  - A. Midazolam
  - B. Propofol
  - C. Haloperidol
  - D. Lorazepam
  - E. Morphine
- 9. A 55-year-old alcoholic male was admitted with status epilepticus and intubated for airway protection. Seizures were initially controlled with bolus doses of lorazepam and loading with phenytoin. He was initially sedated with continuous infusion of lorazepam at 2 mg/h but required increasing doses (10-12 mg/h) by 24 hours, due to manifestations of alcohol withdrawal. After control of seizures, his baseline laboratory results showed sodium 132 mEq/L, chloride 100 mEq/L, potassium 3.4 mEq/L, bicarbonate 22 mEq/L, blood urea nitrogen 22 mg/dL, creatinine 1.5 mg/dL, and glucose 120 mg/dL. Three days into the ICU course, the patient was noted to have a bicarbonate level of 16 mEq/L, with an anion gap of 18, creatinine 1.8 mg/dL, and lactate 2.0 mg/dL. He had an osmol gap of 24 mOsm. He remained sedated, and vital signs were HR 90/min, BP 104/74 mm Hg, RR 12/min (on ventilator), and temperature 98.4° F. Empiric antibiotics were started.

Which one of the following interventions is most appropriate?

- A. Initiate hemodialysis
- B. Stop lorazepam infusion, and start diazepam infusion
- C. Stop lorazepam infusion, and start midazolam infusion
- D. Stop lorazepam infusion, and start fentanyl infusion

10. The morning after an open cholecystectomy a 49-year-old female developed nausea and vomiting. On examination, her abdomen was tympanitic and distended. Diffuse mild pain was experienced with palpation. Radiograph of the abdomen showed normal gas pattern in the small bowel and distended right and transverse colon. Rectal enemas were given, which resulted in a slight decrease in abdominal distension. However, the patient's nausea and vomiting persisted. After several doses of an antiemetic, the patient's rhythm strip showed QT-prolongation and then torsade de pointes. She was successfully resuscitated.

Which one of the following medications was most likely used to treat her nausea and vomiting?

- A. Metoclopramide
- B. Domperidone
- C. Droperidol
- D. Ondansetron
- E. Hydrocortisone
- 11. A 47-year-old female who is 155 cm tall and weighs 100 kg fell down a flight of stairs. Past medical history is positive for insulin-dependent diabetes mellitus for 20 years and chronic alcoholism. Her preoperative blood urea nitrogen was 30 mg/dL, serum creatinine 3.2 mg/dL, serum glucose 160 mg/dL, aspartate aminotransaminase 110 U/L, and international normalized ratio 1.4. After open reduction and internal fixation of a left femoral neck fracture, she is admitted to the ICU because of severe alcoholic withdrawal. The following day, symptoms of withdrawal are much decreased, but she complains of a sudden onset of shortness of breath. A diagnosis of pulmonary embolism is made by helical CT scan. Enoxaparin, 150 mg q 12 h, subcutaneously is begun.

Which one of the following statements about the current enoxaparin dose is most correct?

- A. She is excessively anticoagulated, because dosing should be based on ideal body weight, not actual body weight
- B. She is inadequately anticoagulated, because of morbid obesity and increased volume of distribution
- C. She is excessively anticoagulated, because of hepatic dysfunction
- D. She is inadequately anticoagulated, because of increased cytochrome P450 activity
- E. She is excessively anticoagulated, because of renal dysfunction

## **SECTION 10: PHARMACOLOGY**

**ANSWERS:** 

1-C; 2-D; 3-C; 4-A; 5-B; 6-D; 7-C; 8-A; 9-C; 10-C; 11-E

RATIONALE (1)

Answer: C

Opioids have supraspinal and spinal modes of action. Opioid receptors are found in the central nervous system, especially the midbrain and the lamina I of the dorsal horn in the spinal cord, visceral and vascular smooth muscle, musculoskeletal structures, and at the terminals of peripheral sympathetic and sensory neurons. Opioids modulate pain by (1) presynaptic opioid receptor binding, inhibiting the release of excitatory neurotransmitters; (2) decreased activity in interneurons; and (3) postsynaptic hyperpolarization.

Opioid receptors are classified into groups based on their pharmacologic effects by an agonist. The  $\mu$ -receptors are subdivided into  $\mu_1$ - and  $\mu_2$ -receptors, where the  $\mu_1$ -receptor is responsible for supraspinal analgesia, and the  $\mu_2$ -receptor is responsible for respiratory depression, bradycardia, and physical dependence. Stimulation of delta receptors produces analgesia, mood alterations, and emesis. Kappa receptors modulate spinal analgesia and have minimal respiratory depression. Sigma receptor activation results in excitatory symptoms, such as dysphoria, hypertonia, and tachycardia.

Morphine, meperidine, hydromorphone, and oxycodone are none-specific  $\mu$ -receptor agonists, which mean all will depress respiration to a similar extent when given as equipotent doses. The  $\mu$ -opioid-receptor mediates a dose-dependent depression of ventilation, mainly via a direct action on the medullary respiratory center. A decreased response to hypoxic drive and a decreased response to carbon dioxide occur. Abnormal breathing can be manifested as a shallow, rapid breathing or slow Cheyne-Stokes respiration with normal or large tidal volumes and intermittent apnea. These patients may still be conscious and breathe when reminded.

Butorphanol is an opioid agonist-antagonist. Other agonist-antagonist opioids are nalbuphine and pentazocine. The common effect seen when giving this group of opioids is analgesia with minimal respiratory depression. Each drug has a different effect on the other opioid receptors. Butorphanol does not have significant  $\mu$ -receptor activity while nalbuphine and pentazocine are  $\mu$ -receptor antagonists. Their analgesic activity is modulated by stimulation of spinal  $\kappa$  receptors. Use of this family of opioids on chronic pain patients taking  $\mu$ -receptor agonists, such as oxycodone or methadone, should be avoided or carefully monitored because of the potential for causing opioid withdrawal.

#### REFERENCES (1)

- Austrup ML, Korean G. Analgesic agents for the postoperative period. Surg Clin North Am. 1999; 79:253-273.
- Bowdle TA. Adverse effects of opioid agonists and agonist-antagonists in anaesthesia. *Drug Safety*. 1998; 19:173-189.
- Hoskin PJ, Hanks GW. Opioid agonist-antagonist drugs in acute and chronic pain states. *Drugs*. 1991; 41:326-344.
- Shook JE, Watkins WD, Camporesi EM. Differential roles of opioid receptors in respiration, respiratory disease, and opiate-induced respiratory depression. *Am Rev Respir Dis.* 1990; 142:895-909.
- Schuh KJ, Walsh SL, Stitzer ML. Onset, magnitude and duration of opioid blockade produced by buprenorphine and naltrexone in humans. *Psychopharmacology*. 1999; 145:162-174.

RATIONALE (2)

Answer: D

Propofol has not been causally associated with supraventricular tachycardia in adults when used for ICU sedation. It is known to cause bradycardia, decreased cardiac output, and hypotension as adverse cardiovascular effects. Atrial arrhythmias, ventricular tachycardia, and cardiac arrest have been described, but the causal relationship is not known. Hyperlipemia occurs with an incidence of 3 to 10% and is associated with administration of propofol for an extended period of time. Myoclonic activity versus seizure activity has been described with discontinuation of propofol. Green urine has also been reported with use of propofol. Strict aseptic technique is required for the administration of propofol, due to previously report of bacterial contamination of the product.

### **REFERENCES (2)**

- Angelini G, Ketzler JT, Coursin DB. Use of propofol and other benzodiazepines sedatives in the intensive care unit. Crit Care Clin. 2001; 17:863-880.
- Blakey SA, Hixson-Wallace JA. Clinical significance of rare and benign side effects: propofol and green urine. *Pharmacotherapy.* 2000; 20:1120-1122.
- Medical Economics Company, Incorporated. *Physician's Desk Reference*. 56th ed. Montvale, NJ: Medical Economics Company, Incorporated; 2002, 667-673.

RATIONALE (3)

Answer: C

Hydralazine, because it is associated with reflex tachycardia, is contraindicated in dissecting aortic aneurysm. The associated increase in pulse rate would lead to an increase in the dP/dT ratio, which is the shearing force driving further dissection of the aortic aneurysm. The dP/dT is the change in pressure over change in time and is minimized by keeping pulse low and blood pressure low. Hydralazine's associated reflex tachycardia is also problematic in patients with acute myocardial ischemia or infarction and, in these conditions, is also not a good choice for treating elevated BP. Labetalol is contraindicated in heart blocks greater then first degree but not in first-degree heart block. Labetalol is contraindicated in the presence of significance systolic dysfunction, but, in the patient with diastolic left ventricular dysfunction, the β-blocker effect may be beneficial. Labetalol is a very good drug to treat patients with typical diastolic dysfunction and the associated increased ejection fraction, which is often present. Phentolamine is the drug of choice to treat pheochromocytoma.

#### **REFERENCES (3)**

Cressman MD, Vidt DO, Gifford RW, et al. Intravenous labetalol in the management of severe hypertension and hypertensive emergencies. *Am Heart J.* 1984; 107:980-985.

McKillion PC, Dellinger RP: Hypertensive Emergencies and Urgencies. In: *Critical Care*. 3rd ed. 1997:1811-1822.

Rosenthal T, Rabinowitz B, Boichis H. Use of labetalol in hypertensive patients during discontinuation of clonidine therapy. Eur J Clin Pharmacol. 1981; 20:237-240.

Wright JT, Wilson DJ, Goodman RP, et al. Labetalol by continuous intravenous infusion in severe hypertension. *J Clin Hypertension*. 1986; 1:39-43.

RATIONALE (4)

Answer: A

This patient is developing signs and symptoms consistent with cyanide toxicity from nitroprusside. Nitroprusside has been demonstrated to cause toxicity through the release of cyanide and accumulation of thiocyanate. Cyanide toxicity can present with unexplained cardiac arrest and changes in mental status, including convulsions, encephalopathy, and coma. Metabolic acidosis can also be present, although this may be a late event. Risk of cyanide toxicity can be decreased by utilizing nitroprusside at recommended doses for a short period of time. It has also been recommended that patients receiving high doses of nitroprusside (4-10 mg/kg/min) receive an infusion of thiosulfate. Furthermore, hydroxocobalamin (vitamin 12A) is safe and effective in preventing and treating cyanide toxicity associated with use of nitroprusside. Hydroxocobalamin may be given as a continuous infusion at a rate of 25 mg/h. It is important to note that cyanocobalamin (vitamin B12) is not effective as an antidote and is not capable of preventing cyanide toxicity.

#### **REFERENCES (4)**

Curry SC. Sodium nitroprusside. In: Brent H, et al. Critical Care Toxicology. Philadelphia, PA: Mosby; 2005.

Schulz V, Gross R, Pasch T, et al. Cyanide toxicity of sodium nitroprusside in therapeutic use with and without sodium thiosulphate. Klin Wochenschr 1982; 60:1393-1400.

Varon J, Marik PE. The diagnosis and management of hypertensive crises. Chest. 2000; 118:214-227.

RATIONALE (5)

Answer: B

The utilization of vasopressin in patients with septic shock has been the subject of multiple studies over the last 5 years. It has been reported that endogenous vasopressin levels in patients with septic shock seem to decrease over time and are relatively inadequate for the expected physiologic response, when compared with other types of shock patients. Small trials have demonstrated that the infusion of low-dose vasopressin will restore BP and decrease the use of other catecholamines in patients with septic shock. After initial enthusiasm with these results, caution has been recommended secondary to possible effects of vasopressin on splanchnic circulation, cardiac performance, and unclear outcome data. Vasopressin, when utilized, should be used as hormonal replacement and should not be titrated. Furthermore, it is only recommended that it be used in a dose range of 0.01 to 0.04 U/min (to prevent potential side effects). Vasopressin does not increase cardiac performance (cardiac output/cardiac index), it but has significant potential to impair it. There have been no studies that have demonstrated improved outcomes with the use of vasopressin and patients with septic shock. Recent studies have demonstrated the infusion of vasopressin will decrease index and will produce an increase in the Pco2 gap between the gastric mucosa and arterial blood, suggesting possible deleterious effects on the splanchnic circulation. There is an on going randomized trial evaluating the use of vasopressin and patients with septic shock that may provide further outcome data. Until these results are available, current recommendations are to use vasopressin at a low dose, as cited above, and only in patients with refractory septic shock in which other vasopressors have been used without success.

### **REFERENCES (5)**

Dellinger RP, Carlet JM, Masure H, et al. Surviving Sepsis Campaign Management Guidelines Committee: Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med.* 2004; 32:858-873.

Hollenberg SM, Ahrens TS, Annane D, et al. Practice parameters for hemodynamic support of sepsis in adult patients. Crit Care Med. 2004; 32:1928-1948.

Klinzing S, et al. High-dose vasopressin is not superior to norepinephrine in septic shock. *Crit Care Med.* 2003; 31:2646-2650.

Van Haren FM, et al. The effect of vasopressin on gastric perfusion in catecholamine-dependent patients in septic shock. *Chest.* 2003; 124:2256-2260.

RATIONALE (6) Answer: D

Tigecycline (tygacil<sup>pm</sup>) is a glycylcycline antibacterial drug for IV infusion. Tigecycline is a broad-spectrum antibiotic that has been approved for treatment of infections caused by Grampositive bacteria, Gram-negative bacteria, and anaerobic bacteria. Current clinical indications include complicated skin infections caused by *Escherichia coli*, *Enterococcus faecalis* (vancomycinsusceptible isolates only), *Staphylococcus aureus* (MSSA and MRSA), streptococcus, and *Bacteroides fragilis*. Tigecycline is also indicated for complicated intraabdominal infections caused by resistant Gram-negative bacteria, Gram-positive bacteria, and anaerobic bacteria. Bacteria against which tigecycline is found to have *in vivo* activity for intraabdominal infections include the following: *Citrobacter*, *Enterobacter*, *E coli*, *Klebsiella*, and *E faecalis* (vancomycin-susceptible isolates only), *S aureus* (MSSA only), *Streptococcus*, bacteroides, clostridium perfringens, and *Peptostreptococcus*. Clinical indications are based on clinical trials and *in vivo* testing. However, *in vivo* testing also supports the activity of tigecycline against other organisms, including vancomycin-resistant isolates of *E faecalis*, *Listeria*, *Acinetobacter baumannii*, *Pasteurella multocida*, and *Stenotrophomonas maltophilia*.

#### **REFERENCES (6)**

Clinical and Laboratory Standards Institute (CLSI) [formerly National Committee for Clinical Laboratory Standards (NCCLS)]. Performance standards for antimicrobial susceptibility testing: 15th information supplement. Approved Standard, CLSI document M100-S15, Vol. 25. CLSI, Wayne, PA: CLSI; 2005.

Garrison MW, Neumiller JJ, Setter SM. Tigecycline: an investigational glycylcycline antimicrobial with activity against resistant Gram-positive organisms. *Clin Ther.* 2005; 27:12-22.

RATIONALE (7)

Answer: C

Recombinant human activated protein-C (rh-APC) was approved by the Food and Drug Administration as a therapy for patients with severe sepsis and high risk of death, based on the results of the PROWESS study. The PROWESS study demonstrated a statically significant benefit in those patients who received rh-APC as a continuous infusion for 96 hours, when compared with placebo. This drug has strong anticoagulant effects, as well as antiinflammatory effects, that are thought to be beneficial in modulating the host response seen with severe sepsis. The main complication of this therapy is serious bleeding. It is important for clinicians to understand the potential for bleeding and recognize strategies to minimize this risk when using rh-APC. Practical recommendations to decrease the incident of serious bleeding include discontinuation of the drug for 2 hours prior to invasive procedures and resumption of the infusion 2 hours after the procedure is completed. In this question, the patient has what seems to be an infected central venous catheter that needs to be removed and replaced by a new central venous catheter. Although the platelet count is mildly decreased, it is at a level that traditionally is considered safe for invasive procedures, such as a central venous catheter. Of the answers offered, the one that is most consistent with current recommendations is C, holding the drug for 2 hours, placing the new central catheter in a site that is the left internal jugular vein, and restarting the drug 2 hours after the procedure. There is no need to administer blood products prior to changing prior to the procedure if the drug is discontinued for 2 hours. In addition, changing the line over a guide wire (although it might decrease the incident of mechanical complications and bleeding) is not indicated in this patient because of the clinical findings suggesting an infection of the central venous catheter site.

#### **REFERENCES (7)**

- Bernard GR. Drotrecogin alfa (activated) recombinant human activated protein C for the treatment of severe sepsis. Crit Care Med. 2003; 31:S85-S93.
- Bernard GR, Vincent JL, Laterre PR, et al. Efficacy and safety of recombinant human activated protein c from severe sepsis. *N Engl J Med*. 2001; 344:699-709.
- Fourrier, F. Recombinant human activated protein C in the treatment of severe sepsis: an evidence-based review. *Crit Care Med.* 2004; 11:S534-41.

RATIONALE (8)

Answer: A

With the wide-spread use of antiretroviral therapy in patients with HIV, it is important for the intensivist to recognize some of the potential drug interactions and side effects of antiretroviral medications. Decisions regarding continuation of antiretroviral therapy in patients admitted to the ICU should be discussed with HIV experts. In addition, a team approach with consultation with a critical care pharmacy specialist is important to avoid complications related to drug interactions. Common ICU drugs contraindicated with the use of non-nucleoside reverse-transcriptase inhibitors include midazolam and triazolam. Common ICU drugs, contraindicated with the use of protease inhibitors include the following: midazolam, triazolam, amiodarone, proton-pump inhibitors (with atazanavir), histamine-2 blockers, propafenone, and quinidine. Recognition of potential interaction between these medications and retrovirals is important when treating patients with HIV in the ICU. In this case, of the given list, midazolam would be contraindicated both with the protease inhibitor and the non-nucleoside reverse-transcriptase inhibitors. In such a patient, alternatives to midazolam, such as lorazepam or oxazepam, should be considered.

#### **REFERENCES (8)**

Department of Health and Human Sevices. Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents. Available at: www.aidsinfo.nih.gov/Guidelines/GuidelineDetail.aspx?MenuItem=Guidelines&Search=Off&GuidelineID=7&ClassID. Accessed June 14, 2007.

Huang L, Quartin A, Jones D, et al. Intensive care of patients with HIV infection. N Engl J Med 2006; 355:173-181.

RATIONALE (9)

Answer: C

This clinical situation and the blood chemistry findings in this patient suggest propylene glycol toxicity. Each vial of lorazepam (2 mg/mL) contains 830 mg/mL propylene glycol. The most common manifestations of propylene glycol accumulation are anion gap metabolic acidosis and increased osmol gap. Approximately 12 to 45% of propylene glycol is excreted unchanged in the urine in healthy individuals, and the remainder is metabolized by the liver to lactate and pyruvate. Accumulation may occur when doses exceed the upper recommended dose of 0.1 mg/kg/h. Renal and/or hepatic insufficiency may also play a role in accumulation. Serious reported toxicities include renal dysfunction, hemolysis, cardiac arrhythmias, seizures and central nervous system depression, or agitation. The metabolic findings of propylene glycol toxicity may also be confused with sepsis or severe inflammatory states.

Clinical studies suggest that the osmol gap correlates with propylene glycol accumulation. Although toxicity is more common after long periods of lorazepam infusion (>3 days), toxicity has occurred with short-term and high-dose use. The treatment of choice is to stop the lorazepam infusion and sedate with an agent that does not contain propylene glycol. Diazepam contains propylene glycol, but midazolam does not. It would not be appropriate to change to fentanyl alone in a patient who requires benzodiazepines for alcohol withdrawal. Hemodialysis would remove propylene glycol but is not required unless severe renal dysfunction develops. There is a case report of propylene glycol toxicity developing in a patient receiving continuous renal replacement. Propylene glycol is metabolized by alcohol dehydrogenase, but there are no reports of using ethanol infusion or fomepizole for toxicity.

#### **REFERENCES (9)**

Arroliga AC, Shehab N, McCarthy K, Gonzales JP. Relationship of continuous infusion lorazepam to serum propylene glycol concentration in critically ill adults. Crit Care Med. 2005; 32:1709-1714.

Parker MG, Fraser GL, Watson DM, Riker RR. Removal of propylene glycol and correction of increased osmolar gap by hemodialysis in a patient on high dose lorazepam infusion therapy. *Intensive Care Med.* 2002; 28:81-84.

Wilson KC, Reardon C, Theodore AC, Farber HW. Propylene glycol toxicity: a severe iatrogenic illness in ICU patients receiving IV benzodiazepines. *Chest.* 2005; 128:1674-1681.

Yaucher NE, Fish JT, Smith HW, Wells JA. Propylene glycol-associated renal toxicity from lorazepam infusion. *Pharmacotherapy*. 2003; 23:1094-1099.

RATIONALE (10)

Answer: C

A number of drugs can lead to QT prolongation and torsade de pointes. Droperidol, a butyrophenone derivative, is an antiemetic that has the potential of prolonging the QT interval. Fortunately, it rarely produces this phenomenon at recommended doses. Antiemetics, such as ondansetron (serotonin antagonists), metoclopramide (antidopaminergic and antiserotinergic), and dronabinol (cannabinoid derivative), are not known to cause QT prolongation. Phenothiazines used for nausea and vomiting may also cause QT prolongation but is not one of the answer choices. Targeted drug therapy for nausea and vomiting can improve the success of relieving symptoms. In addition, being aware of associated adverse drug reactions can help decrease the drug-related complications from the drug itself or through drug-drug interactions.

Postoperative nausea and vomiting are often multifactorial in origin. Drugs, physical stimuli, or emotional stress can cause the release of neurotransmitters that stimulate serotoninergic (5-HT<sub>3</sub>), dopaminergic (D<sub>2</sub>), histaminergic (H<sub>1</sub>), and muscarinic (M<sub>1</sub>) receptors. The receptor stimulation in the chemoreceptor trigger zone, gastrointestinal tract, vestibular apparatus, pharynx, or cerebral cortex triggers neurogenic signals to be sent to the vomiting center in the brainstem. The vomiting center, rather than being a discrete area, is more of a neural network comprised of the chemoreceptor trigger zone, area postrema, and nucleus tractus solitarius.

Vagal afferent signals through the nodosum ganglion and the nucleus tractus solitarius mediate nausea that arises from gastric irritants; gastric, small intestinal, colonic, or bile duct distention; and inflammation or ischemia of bowel, liver, pancreas, and peritoneum.

Phenothiazines and butyrophenones act on D<sub>2</sub>, H<sub>1</sub>, and M<sub>1</sub> receptors. Benzamides, such as metoclopramide and domperidone, affect 5-HT<sub>3</sub> and 5-HT<sub>4</sub> receptors; scopolamine is an M<sub>1</sub>-receptor antagonist; and diphenhydramine and cyclizine are H<sub>1</sub>-antagonists. Specific 5-HT<sub>3</sub>-receptor antagonists, such as ondansetron and granisetron, are the most recently developed class of antiemetics.

#### **REFERENCES (10)**

De Ponti F, Poluzzi E, Montanaro N. Organising evidence on QT prolongation and occurrence of Torsades de Pointes with nonantiarrhythmic drugs: a call for consensus. *Eur J Clin Pharmacol.* 2001; 57:185-209.

Drolet B, Zhang S, Deschenes D, et al. Droperidol lengthens cardiac repolarization due to block of the rapid component of the delayed rectifier potassium current. *J Cardiovasc Electrophysiol*. 1999; 10:1597-1604.

Michalets EL, Smith LK, Van Tassel ED. Torsade de pointes resulting from the addition of droperidol to an existing cytochrome P450 drug interaction. *Ann Pharmacother*. 1998; 32:761-765.
Sung YF. Risks and benefits of drugs used in the management of postoperative nausea and vomiting. *Drug Safety*. 1996; 14:181-197.

RATIONALE (11)

Answer: E

Low-molecular-weight heparins (LMWHs) do not undergo hepatic metabolism and primarily undergo renal elimination. Generally, dosing of LMWH is based on actual body weight, however, if a patient weighs more than 130 kg, using standard dosing may cause excessive anticoagulation. This patient is likely to be overanticoagulted from LMWH accumulation due to her renal failure, not from excessive LMWH dose.

The primary advantages of LMWH, compared with unfractioned heparin (UFH), are better bioavailability and consistency of action. Bioavailability is greater than 90% for LMWHs, but only 30% for UFH. This difference is related to UFH's nonspecific binding to proteins and cells. This binding contributes not only to its poor bioavailability but also effects anticoagulant activity and decreases plasma half-life. UFH is metabolized by a rapid, zero-order kinetic enzyme system within the liver. The saturable hepatic kinetics results in inconsistent elimination that also increases variability in drug effect. UFH and its metabolites undergo first-order renal clearance kinetics. In contrast, less-negative-charged LMWH molecules result in relatively little nonspecific protein binding, and dose-independent renal clearance of LMWHs result in predictable antithrombotic activity, and, typically, anticoagulation monitoring is not needed.

Plasma half-life of LMWHs is approximately 2- to 4-fold longer than that of heparin, enabling them to be administered only once or twice daily. Subcutaneous LMWHs are well absorbed from the subcutaneous tissue, are rapidly distributed to most organs and tissues, and attain antithrombotic levels within 30 minutes of administration. Subcutaneous absorption of heparin is more variable, however, and antithrombotic levels might not be reached for 1 to 2 hours.

Anticoagulation with LMWH can be achieved with minimal elevation of partial thromboplastin time. When monitoring is done, synthetic substrate-based assays for measuring thrombin and factor Xa inhibition are used. The anti-Xa assays are more sensitive to the effects of LMWHs and are used more commonly than antithrombin assays.

The College of American Pathologists released a consensus report on the laboratory monitoring of anticoagulant therapy in 1998. When monitoring LMWH, the College of American Pathologists recommends checking anti-Xa activity at peak effect (approximately 4 hours after administration), because peak levels correlate with safety and efficacy over trough levels, obtained just prior to administration of a dose. For the acute management of venous thromboembolic disease, the College of American Pathologists suggests a peak target level of 0.5 to 1.1 U/mL for twice-daily recipients and 1.0 to 2.0 U/mL for once-daily patients. The College of American Pathologists recommends monitoring for patients who have renal insufficiency, for patients who will be receiving LMWHs for prolonged periods (months), or for patients who are at extreme body weights (130 kg) or who are newborn.

#### **REFERENCES (11)**

- Hirsh J, Warkentin TE, Shaughnessy SG, et al. Heparin and low-molecular-weight heparin: Mechanisms of action, pharmacokinetics, dosing, monitoring, efficacy, and safety. *Chest.* 2001; 119:64S-94S.
- Mousa SA. Comparative efficacy of different low-molecular-weight heparins (LMWHs) and drug interactions with LMWH: implications for management of vascular disorders. Semin Thromb Hemost. 2000; 26:39-46.
- Samama MM, Gerotziafas GT. Comparative pharmacokinetics of LMWHs. Semin Thromb Hemost. 2000; 26:31-38.
- Smith BS, Gandhi PJ Pharmacokinetics and pharmacodynamics of low-molecular-weight heparins and glycoprotein IIb/IIIa receptor antagonists in renal failure. *J Throm Thrombolysis*. 2001; 11:39-48.
- Spencer FA, Ball SP, Zhang Q, Liu L, Benoit S, Becker RC. Enoxaparin, a low-molecular-weight heparin, inhibits platelet-dependent prothrombinase assembly and activity by factor-Xa neutralization. *J Thromb Thrombolysis*. 2000; 9:223-228.

# SECTION 11: Poisoning/Overdose

# SECTION 11: POISONING/OVERDOSE

Instructions: For each question, select the most correct answer.

1. A 35-year-old man is admitted to the ICU with a reported history of ethylene glycol ingestion. A friend found him comatose after not being able to contact him for 48 hours. An empty can of antifreeze was found in the apartment. The patient is intubated for airway protection. Vital signs include BP 110/60 mm Hg, HR 110/min, RR 18/min, afebrile. Physical examination is remarkable for obtundation, tachycardia, and bilateral diffuse rales. Laboratory examination revealed a room air arterial blood gas measurement with pH 7.09, Paco<sub>2</sub> 30 mm Hg, Pao<sub>2</sub> 65 mm Hg, an anion gap of 22, a normal osmolar gap, creatinine 2.4 mg/dL, and negative serum ethanol. An ethylene glycol level is ordered, but results will not be available for 72 hours.

Which one of the following interventions is most likely to benefit this patient?

- A. Ethanol infusion
- B. Fomepizole IV
- C. Hemodialysis
- D. Thiamine and pyridoxine IV
- E. IV crystalloids

2. A 30-year-old man ingested 30 tablets of phenytoin 100 mg, 20 tablets of ibuprofen 200 mg, and 10 tablets of amoxicillin 500 mg 3 hours ago. He is lethargic but arouses with verbal and tactile stimulation.

Which one of the following is the most appropriate method of gastric decontamination?

- A. Sorbitol cathartic
- B. Whole bowel irrigation
- C. Activated charcoal
- D. Intubation and gastric lavage
- E. Gastric lavage and activated charcoal

3. A 35-year-old woman with an anxiety disorder ingested 20 tablets of alprazolam 0.1 mg and 5 carisoprodol, along with alcohol, in a suicide attempt approximately 5 hours prior to arrival. In the emergency department, she is arousable only to sternal rub. Vital signs include BP 100/60 mm Hg, pulse 68/min, RR 8/min, temperature 36.0°C (96.8°F).

Which one of the following interventions is most appropriate for her management?

- A. Intubation
- B. Intubation and gastric lavage
- C. Flumazenil IV
- D. Observation
- E. Administration of oral activated charcoal
- 4. Three intubated patients are admitted to the ICU following exposure to an unknown gas in a bus station. Clinical findings include severe hypoxemia with copious respiratory secretions and wheezing. Additional findings include small pupils, bradycardia, and diarrhea. One of the patients is able to follow commands but appears to have significant diffuse weakness.

Which one of the following interventions would be most beneficial?

- A. Pyridostigmine
- B. Atropine
- C. Sodium nitrite and sodium thiosulfate
- D. Hyperbaric oxygen
- E. Pralidoxime

5. A 17-year-old female is brought to the hospital after ingesting approximately 25 tablets of acetaminophen (325 mg) and an unknown amount of verapamil. The ingestion occurred approximately 4 hours prior to arrival. Initial vital signs revealed BP 104/37 mm Hg, HR 119/min, and respirations 20/min, afebrile. The patient received oral charcoal. Her initial electrocardiograph is shown in Figure 1. Approximately 1 hour later, the patient is noted to have decreased mental status, BP 76/32 mm Hg, and HR 60/min. The electrocardiograph at this time is illustrated in Figure 2.

Which one of the following sequence of interventions is the most appropriate to stabilize the patient assuming that the preceding intervention is unsuccessful?

- A. Transthoracic pacing, then transvenous pacing
- B. IV glucagon, then transthoracic pacing, then transvenous pacing
- C. IV glucagon, then IV calcium chloride, then transthoracic pacing
- D. IV calcium chloride, then transthoracic pacing
- E. IV calcium chloride, then IV glucagon, then transthoracic pacing

Figure 1

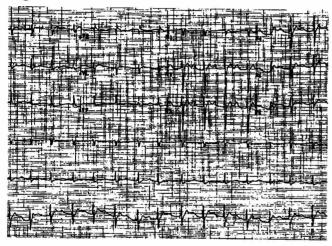
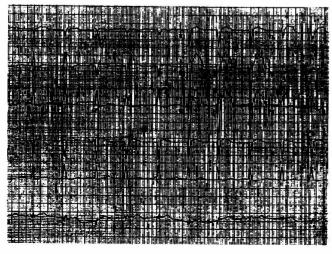


Figure 2



6. An obtunded patient is intubated for an overdose of amitriptyline. The electrocardiogram reveals a QRS of 0.14 seconds, and sodium bicarbonate was administered to achieve a blood pH of 7.50. Despite large volumes of IV fluids, the BP remains 80/40 mm Hg.

Which one of the following interventions is indicated?

- A. More sodium bicarbonate
- B. Dopamine
- C. Physostigmine
- D. Norepinephrine
- E. Magnesium sulfate
- 7. A 50 year-old homeless man is found unconscious outside the police station. His fingerstick glucose by paramedics was 20 mg/dL. He awakens with 50% glucose. On arrival to the hospital, his vital signs are BP 160/94 mm Hg, HR 90/min, RR 12/min, and temperature 36.1°C (97.0°F). Physical examination is unremarkable. The patient denies having diabetes or using insulin but confirms a history of hypertension. During his evaluation, the patient becomes obtunded and a fingerstick glucose is 18 mg/dL. After an initial response to 50% glucose, he becomes unresponsive and hypoglycemic 20 minutes later. The patient is admitted to the ICU and is intubated to protect his airway. An IV infusion of 10% glucose is started, but the patient has multiple episodes of hypoglycemia requiring bolus doses of 50% glucose.

Which one of the following interventions is most likely to be of benefit in this patient?

- A. Administration of subcutaneous octreotide
- B. Administration of 20% glucose by peripheral IV
- C. Administration of 50% glucose by nasogastric tube
- D. Initiation of hemodialysis
- E. Administration of intramuscular glucagon

8. 35 year-old woman is found in her car with the ignition on and is brought to the hospital. She is unresponsive and intubated for airway protection. Vital signs are BP 96/54 mm Hg, HR 115/min, RR 12/min, and temperature 36.1°C (97.0°F). Physical examination is remarkable for obtundation, tachycardia, and bibasilar rales. Pupils are 3 mm and sluggishly reactive. Laboratory examination revealed an arterial blood gas with pH 7.27, Paco<sub>2</sub> 35 mm Hg, PaO<sub>2</sub> 365 mm Hg (while receiving 100% O<sub>2</sub>), an anion gap of 18 mmol/L, normal osmolar gap, and negative serum ethanol. A carboxyhemoglobin level is 15%. An ECG shows nonspecific ST segment changes. Salicylate and acetaminophen levels are pending.

Which one of the following interventions is most appropriate for this patient at this time?

- A. Administer flumazenil
- B. Administer naloxone
- C. Hemodialysis
- D. Continue 100% oxygen
- E. Administer hyperbaric oxygen
- 9. An intubated ICU patient has overdosed on a monoamine oxidase inhibitor and fluoxetine. The patient is delirious, has had several seizures, and has tremors of the extremities.

Which one of the following should be administered?

- A. Dantrolene
- B. Lorazepam
- C. Propranolol
- D. Bromocriptine
- E. Neuromuscular blocker

10. An obtunded patient with bipolar disorder is admitted to the ICU with a lithium level of 4.2 mmol/L (therapeutic range 0.6-1.2 mEq/L). The patient was chronically treated with lithium but had ingested a large number of sustained-released lithium 6 hours prior to admission. Hemodialysis was instituted and the lithium level decreased to 1.0 mmol/L, but the patient remained obtunded.

Which one of the following is the most appropriate intervention at this time?

- A. Further emergent hemodialysis
- B. Normal saline solution diuresis
- C. Administer sodium polystyrene sulphonate
- D. Close observation, and repeat lithium level in 6-8 hours
- E. Administer activated charcoal every 4-6 hours
- 11. Which one of the following statements is true regarding an overdose with valproic acid?
  - A. Hepatotoxicity is common following an acute overdose
  - B. Hyponatremia has been associated with high drug levels
  - C. Multiple dose activated charcoal is recommended for severe toxicity
  - D. A patient with a therapeutic level 2 hours after ingestion may be discharged if there is no evidence of central nervous system depression
  - E. Cerebral edema associated with valproic acid may occur 24-72 hours after ingestion
- 12. A 25 year-old man ingested approximately 30 pills of an extra-strength acetaminophen product (500 mg/pill), 50 ibuprofen tablets (200 mg/pill) and 20 pills of alprazolam (unknown dose) approximately 9 hours prior to presentation in a suicide attempt. In the ICU, he is drowsy but arousable and complains of nausea and vomiting. Vital signs are BP 120/76 mm Hg, HR 90/min, RR 10/min, and temperature 37.0°C (98.6°F).

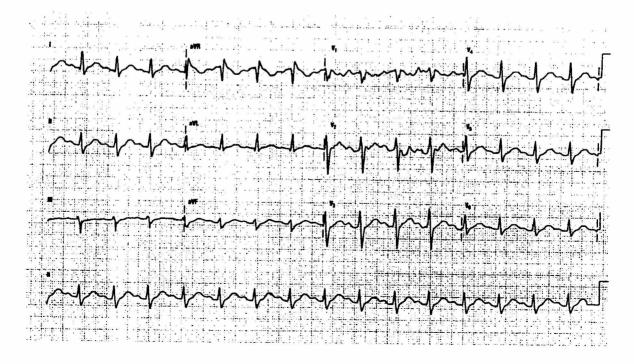
Which one of the following is the most appropriate intervention in addition to obtaining an acetaminophen level?

- A. Obtain liver function tests
- B. Await results of laboratory tests
- C. Administer oral N-acetylcysteine
- D. Administer oral activated charcoal
- E. Administer IV flumazenil

13. A 35-year-old male with suspected drug abuse was found naked running down the freeway. In route to the hospital, he was noted to have a generalized tonic-clonic seizure lasting approximately 3 minutes. On arrival to the hospital, his vital signs were BP170/98 mm Hg, HR 120/min, RR 18/min, and oral temperature 39.3°C (102.8°F). He is noted to be agitated, diaphoretic, and voicing paranoid thoughts. He is restrained and IV fluids are initiated.

Which one of the following should be initially administered to this patient?

- A. Labetalol
- B. Haloperidol
- C. Phenytoin
- D. Lorazepam
- E. Acetaminophen
- 14. A 58-year-old male is brought to the emergency department by his wife after developing altered mental status. The wife states she found an empty bottle of pills at his bedside. The patient is somnolent and confused. An ECG is done and is shown below. Which of the following is the most appropriate intervention at his moment?
  - A. Sodium bicarbonate
  - B. Digoxin Immune Fab
  - C. Calcium chloride
  - D. Naloxone
  - E. Glucagon



# SECTION 11: POISONING/OVERDOSE

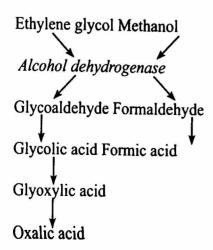
**ANSWERS:** 

1-C; 2-C; 3-A; 4-B; 5-E; 6-D; 7-A; 8-E; 9-B; 10-D; 11-E; 12-C; 13-D; 14-A

RATIONALE (1)

Answer: C

Hemodialysis is most likely to benefit this patient by removing acid metabolites of ethylene glycol. Methanol and ethylene glycol poisonings may be characterized by the presence of an osmolar gap and anion gap metabolic acidosis. All alcohols are osmotically active substances, but only methanol and ethylene glycol are metabolized to acids (see figure below). Isopropyl alcohol is metabolized to acetone and does not result in an anion gap metabolic acidosis. However, patients ingesting ethylene glycol may present with a normal osmolar gap due to complete metabolism of the alcohol to toxic metabolites (late presentation) or errors in the performance of the serum osmolarity. Similarly, patients ingesting ethylene glycol may present with an osmolar gap and normal anion gap, if concurrent ethanol ingestion prevents metabolism to acid metabolites or they present very early after ingestion. Ethanol is preferentially metabolized by alcohol dehydrogenase. In this patient, the history is strongly suggestive of ethylene glycol ingestion, and it is supported by the laboratory findings of anion gap acidosis, obtundation, and renal insufficiency. The low pH is a poor prognostic marker and immediate hemodialysis should be initiated. The value of ethanol infusion or fomepizole in a delayed presentation is questionable, because ethylene glycol is likely to be completely metabolized by alcohol dehydrogenase to toxic metabolites. However, if there is any suspicion of the presence of ethylene glycol, oral, or IV ethanol or IV fomepizole can be initiated. IV fluids are indicated for volume resuscitation, and mechanical ventilatory support can be increased to offset the low pH but provide less benefit than hemodialysis. There are no data to support the effectiveness of thiamine and pyridoxine in ethylene glycol intoxication, but they can be administered to patients with possible deficiencies (eg, alcoholics). Delayed presentations usually do not have urinary oxalate crystals that may be present transiently within 4 to 6 hours after ethylene glycol ingestion.



#### REFERENCES (1)

- Barceloux DG, Bond GR, Krenzelok EP, et al. American Academy of Clinical Toxicology practice guidelines on the treatment of methanol poisoning. *J Toxicol Clin Toxicol* 2002; 40:415-446.
- Barceloux DG, Krenzelok EP, Olson R, Watson W. American Academy of Clinical Toxicology practice guidelines on the treatment of ethylene glycol poisoning. *J Toxicol Clin Toxicol* 1999; 37:537-560.
- Brent J, McMartin K, Phillips S, et al. Fomepizole for the treatment of ethylene glycol poisoning. *N Engl J Med* 1999; 340:832-838.
- Brent J, McMartin K, Phillips S, et al. Fomepizole for the treatment of methanol poisoning. *N Engl J Med* 2001; 344:424-429.
- Glaser DS: Utility of the serum osmol gap in the diagnosis of methanol or ethylene glycol ingestion. Ann Emerg Med 1996; 27:343-346.
- Megarbane B, Borron SW, Baud SJ. Current recommendations for treatment of severe toxic alcohol poisonings. *Intensive Care Med* 2005;31:189-195.

RATIONALE (2)

Answer: C

There is little evidence of benefit for any method of gastric decontamination in overdose victims, but, in this patient, activated charcoal is the best response. Activated charcoal adsorbs most ingested drugs and is likely to be more effective if given early. Cathartics have been used for many years in poisonings based on the hypothesis that pushing the drug through the gastrointestinal tract faster will decrease absorption. No evidence exists to support that premise, and cathartics are not routinely recommended. Whole bowel irrigation is not specifically indicated for any poisoning but may be considered in overdoses with agents that are not adsorbed by charcoal (iron, lithium), sustained-release drugs, and illicit drug packets. Volunteer studies have raised concerns about the efficacy of eliminating drugs using this method. Whole bowel irrigation would not be considered in this patient given the drugs that were ingested. Gastric lavage is not indicated in this patient. There is no known benefit of gastric lavage, even when used within 1 hour of ingestion. The procedure is associated with risks of aspiration and esophageal perforation. In this patient, the ingestion is not life-threatening, and most drugs would no longer be in the stomach at 3 hours. Current recommendations suggest that gastric lavage can be considered in life-threatening overdoses, if they present within 1 hour of ingestion. Ipecac is no longer recommended for gastric decontamination and should not be used.

#### **REFERENCES (2)**

American Academy of Clinical Toxicology; European Association of Poison Centres and Clinical Toxicologists. Position paper: Cathartics. *J Toxicol Clin Toxicol* 2004; 42:243-253.

American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Position paper: Gastric lavage. *J Toxicol Clin Tox*icol 2004; 42:933-943.

American Academy of Clinical Toxicology; European Association of Poison Centres and Clinical Toxicologists. Position paper: Single-dose activated charcoal. *J Toxicol Clin Toxicol* 2005; 43:61-87.

American Academy of Clinical Toxicology; European Association of Poison Centres and Clinical Toxicologists. Position paper: Whole bowel irrigation. *J Toxicol Clin Toxicol* 2004;42:843-854.

RATIONALE (3)

Answer: A

This patient has ingested several sedating drugs that contribute to her clinical presentation. The best intervention for this patient is intubation to protect the airway and supportive care until awakening. Activated charcoal is unlikely to be of any benefit due to the delay in presentation. Drug is unlikely to be present in the upper gastrointestinal tract. Likewise, gastric lavage is not warranted 5 hours after ingestion. Observation alone is also inappropriate due to her severe depressed consciousness and the need to protect her airway from aspiration. Although flumazenil is an antidote for benzodiazepines, it would be contraindicated in this patient who is likely chronically ingesting alprazolam for her anxiety disorder. The risk of administering flumazenil includes benzodiazepine withdrawal seizures, which would be extremely difficult to treat, because benzodiazepine receptors would be blocked. Flumazenil administration is also contraindicated in patients who may have ingested cyclic antidepressants due to the risk of seizures. Another disadvantage of flumazenil is the short half-life, which may result in resedation after patient arousal. Flumazenil administration may be considered as a diagnostic tool in an obtunded patient with no potential contraindications but should not be used as an alternative to other methods of airway protection.

Benzodiazepines are usually low-risk for death in overdose patients, unless other sedating drugs are involved, as in this patient. There is some suggestion that alprazolam may be more toxic and result in longer ICU stays and greater need for intubation. Alprazolam is also a commonly abused drug.

#### **REFERENCES (3)**

Isbister GK, O'Regan L, Sibbitt D, Whyte IM. Alprazolam is relatively more toxic than other benzodiazepines in overdose. *Br J Clin Pharmacol* 2004;58:88-95.Seger DL. Flumazenil-treatment or toxin. *J Toxicol Clin Toxicol* 2004;42:209-216.

RATIONALE (4)

Answer: B

Poisoning with nerve gases, such as sarin (O-Isopropyl methylphosphonofluoridate), result in a cholinergic syndrome suggested by the clinical findings. A cholinergic syndrome can also result from organophosphate or carbamate poisoning. The toxicity is caused by inhibition of cholinesterase. with no degradation of acetylcholine at the postsynaptic receptor. Muscarinic effects include bronchorrhea, bradycardia, and a hypersecretory syndrome (salivation, lacrimation, urination, defecation, emesis). Nicotinic effects result in muscle fasciculations and weakness, and central nervous system effects lead to headache, confusion, and central respiratory depression. Pupils are typically mioitic. The primary concern in such patients is hypoxic respiratory failure from bronchorrhea, bronchospasm, and respiratory depression. IV atropine should be administered in doses of 2 to 4 mgs, repeated every 5 minutes until tracheobronchial secretions are controlled. The most common cause of death in cholinergic poisonings is asphyxiation. Continued administration of large doses of atropine may be required as bolus or continuous infusion. Because sarin irreversibly binds to cholinesterase, the patient is likely to require continuous atropine infusion in an ICU setting until the agent is completely metabolized. Atropine does not reverse nicotinic effects and pralidoxime (2-PAM) is used to reverse muscle weakness by regeneration of acetylcholinesterase. It is administered as a loading dose (1 to 2 gm in 500 mL normal saline solution over 30 minutes) and then as a continuous infusion at 500 mg/h. Pyridostigmine, an anticholinesterase, may precipitate a cholinergic crisis and is used for treatment of myasthenia gravis. Hyperbaric oxygen may be considered for patients with severe carbon monoxide poisoning, but patients would not typically have evidence of a hypersecretory syndrome. Patients with carbon monoxide poisoning will often present comatose with an anion gap metabolic acidosis, cardiovascular instability and increased levels of carboxyhemoglobin. Sodium nitrite and sodium thiosulfate are used in antidotal treatment of cyanide poisoning. Cyanide poisoning usually presents with more life-threatening manifestations, such as sudden cardiovascular collapse, seizures, hypotension, arrhythmias, and severe metabolic acidosis.

#### **REFERENCES (4)**

- Bardin PG, Van Eden SF, Moolman JA, et al. Organophosphate and carbamate poisonings. *Arch Intern Med* 1994; 154:1433-1441.
- Center for Disease Control. Sarin (GB). Available at: www.bt.cdc.gov/agent/sarin/. Accessed June 13, 2007.
- Leiken JB, Thomas RJ, Walter FG, et al. A review of nerve agent exposure for the critical care physician. Crit Care Med 2002;30:2346-2354.
- Peter JV, Cherrian AM. Organic insecticides. Anaes Intens Care 2000; 28:11-21.
- Tush GM, Anstead MI. Pralidoxime continuous infusion and the treatment of organophosphate poisonings. *Ann Pharmacother* 1997; 31:441-444.

RATIONALE (5)

Answer: E

The appropriate initial intervention to stabilize this patient with hypotension and bradycardia secondary to ingestion of a calcium-channel blocker is the administration of IV calcium chloride. Calcium is effective in reversing toxic effects in approximately 50% of patients. The next intervention is IV glucagon and, subsequently, transthoracic pacing, if other interventions fail. The hypotension in calcium-channel blocker overdose, especially verapamil, is significantly related to negative inotropy. Glucagon acts as an inotropic agent by increasing intracellular cyclic AMP similar to adrenergic agents. Transthoracic pacing may increase the heart rate but not necessarily improve the BP. In this patient, her HR of 60/min should have been adequate to maintain her BP, if myocardial function was normal. Because most calcium channel blockers are sustainedreleased preparations, interventions may need to be continued for 24 to 48 hours. Infusions of calcium (high doses may be required) can be used, as well as IV infusions of glucagon. If significant bradycardia develops, transthoracic or transvenous pacing may be needed. Atropine and other adrenergic drugs are often ineffective in these overdoses. Additional interventions that can be considered include insulin euglycemia that involves the administration of insulin that acts as an inotropic agent and glucose infusion to counteract potential hypoglycemia. Experience is limited with this intervention. In severe toxicity, a phosphodiesterase inhibitor, such as milrinone, can be considered. However, use of these agents may worsen hypotension. Cardiopulmonary bypass has also been reported to sustain hemodynamic status in refractory cases.

#### **REFERENCES (5)**

- Bailey B. Glucagon in β-blocker and calcium channel blocker overdoses: a systematic review. *J Toxical Clin Toxicol* 2003;41:595-602.
- DeRoos F. Calcium channel blockers. In: Goldfrank's Toxicologic Emergencies. 6th ed. Goldfrank LR, Flomenbaum NE, Lewin NA, et al, eds. Stamford, CT: Appleton & Lange, 1998, 829-843.
- Lam Y, Tse H, Lau C. Continuous calcium chloride infusion for massive nifedipine overdose. *Chest* 2001; 119:1280-1282.
- Yuan TH, Kerns WP, Tomaszewski CA, et al. Insulin-glucose as adjunctive therapy for severe calcium channel antagonist poisoning. *J Toxicol Clin Toxicol* 1999; 37:463-474.

RATIONALE (6)

Answer: D

In cyclic antidepressant overdose, hypotension refractory to volume expansion is best treated with a direct-acting adrenergic agent. Cyclic antidepressants result in depletion of norepinephrine stores and a direct α-adrenergic agent, such as norepinephrine or phenylephrine, is indicated to improve BP. Dopamine is less effective in this setting, because it acts by causing release of norepinephrine. The administration of bicarbonate with cyclic antidepressant overdose is most effective for evidence of cardiac toxicity (widened QRS or arrhythmias), rather than hypotension or seizures. The major effect of bicarbonate appears to be increased sodium conductance through myocardial fast sodium channels (sodium loading), rather than an increase in plasma protein binding of free tricyclic medication. Bicarbonate may improve hypotension by reversing myocardial dysfunction. Additional sodium bicarbonate in this patient may lead to volume overload and pulmonary edema. An inotrope, such as dobutamine, should be added only if hypotension is known or suspected to be the result of depressed myocardial contractility with decreased cardiac output. Magnesium sulfate is indicated for the treatment of torsades de pointes due to cyclic antidepressant toxicity and would lower the BP further. Physostigmine may reverse the altered mental status in anticholinergic syndromes, but it is not recommended due to the potential for seizures and asystole. Hypotension refractory to fluid and vasopressors may warrant consideration of an intraaortic balloon pump as a temporizing measure.

#### **REFERENCES (6)**

Buchman AL, Dauer J, Geideman J. The use of vasoactive agents in the treatment of refractory hypotension seen in tricyclic antidepressant overdose. *J Clin Psychopharmacol* 1990; 10:409-413. Glauser J. Tricyclic antidepressant poisoning. *Cleveland Clin J Med* 2000; 67:704-719. Kerr GW, McGuffie AC, Wilkie S. Tricyclic antidepressant overdose: a review. *Emerg Med J* 2001; 18:236-241.

Zimmerman JL, Rudis M. Poisonings. In: Critical Care Medicine. Parrillo JE, Dellinger RP, eds. St. Louis, MO: Mosby, Inc., 2001, 1501-1524.

RATIONALE (7)

Answer: A

This patient's presentation is suggestive of possible overdose with a hypoglycemic agent, such as insulin or sulfonylurea. Severe, prolonged hypoglycemia poorly responsive to IV glucose is unusual with insulin. However, it is characteristic of ingestion of large doses of sulfonylureas. Sulfonylureas stimulate insulin release from the pancreas, resulting in hypoglycemia. Insulin and C-peptide levels may be mildly elevated or high normal in patients who overdose on sulfonylureas. Octreotide is a somatostatin analogue that inhibits release of insulin from the pancreas and has been found to be effective in treating hypoglycemia and shortening the period of hypoglycemia. Variable doses have been used, ranging from single IV or subcutaneous doses of 50-100 µg to multiple doses (50 µg every 8 hours) or IV infusions. Administering glucose can increase the blood glucose but will also induce insulin release. Central venous access is needed for administering concentrated glucose solutions due to hyperosmolarity that can cause endothelial damage. Oral administration of glucose in a severely ill patient is not reliable. Hemodialysis is of no benefit in this situation, because sulfonylureas are protein bound. Although glucagon stimulates hepatic glycogenolysis and can increase glucose levels, this patient is likely to have depleted glycogen stores by this time.

#### **REFERENCES (7)**

Carr R, Zed PJ. Octreotide for sulfonylurea-induced hypoglycemia following overdose. *Ann Pharmacother* 2002;36:1727-1732.

Green RS, Palatnik W. Effectiveness of octreotide in a case of refractory sulfonylurea-induced hypoglycemia. *J Emerg Med* 2003;25:283-287.

RATIONALE (8)

Answer: E

This patient's presentation is suggestive of probable carbon monoxide poisoning for several reasons. She was found in the appropriate setting, the clinical findings are consistent with carbon monoxide toxicity, and the laboratory results also support exposure to carbon monoxide. The best confirmation of significant carbon monoxide exposure is an elevated carboxyhemoglobin level (venous or arterial sample). However, the initial level may not be helpful if a significant period of time has elapsed since exposure. Carboxyhemoglobin levels of up to 10% can be found in people in some urban areas and in heavy smokers. Pulse oximetry often overestimates oxygenation in the setting of carbon monoxide toxicity so it is not helpful in the assessment of patients. The presence of metabolic acidosis suggests tissue hypoxia, and a lactate level is more reliable than the carboxyhemoglobin level in determining the severity of toxicity. Although there is continued debate on the indications for hyperbaric oxygen therapy, most clinicians would agree that the presence of significant neurologic impairment would warrant treatment. Hemoglobin binds carbon monoxide tightly (approximately 240 times greater than for oxygen) and forms a complex that is only slowly reversible. This binding can be overcome by high tissue levels of oxygen. The half-life of carboxyhemoglobin falls from 4 to 6 hours while receiving room air, to approximately 90 minutes while receiving 100% oxygen at 1 atmosphere of pressure, and to approximately 30 minutes while

receiving 100% oxygen at 3 atmospheres of pressure. Although 100% oxygen shortens the half-life of carboxyhemoglobin, a recent study suggests that it is less effective at reducing postexposure cognitive deficits, compared with hyberbaric oxygen. There is no reason to administer flumazenil or naloxone, because the patient is supported by mechanical ventilation. There is no indication for dialysis in this patient.

#### **REFERENCES (8)**

Domachevsky L, Adir Y, Grupper M, Keynan Y. Hyperbaric oxygen in the treatment of carbon monoxide poisoning. *J Toxicol Clin Toxicol* 2005;43:181-188.

Juurlink DN, Buckley NA, Stanbrook MB, et al. Hyperbaric oxygen for carbon monoxide poisoning. *Cochrane Database Syst Rev* 2005 Jan 25;(1):CD002041.

Weaver LK, Hopkins RO, Chan KJ, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med 2002;347:1057-1067.

RATIONALE (9)

Answer: B

The patient described is suffering from selective serotonin reuptake inhibitor (SSRI) syndrome. The syndrome may occur after a single dose, high dose overdose or when combined with other serotonergic agents. The pathophysiology is probably related to excessive stimulation of serotonin receptors in the brain. Clinical manifestations include altered mental status, autonomic dysfunction, and neuromuscular abnormalities ranging from tremors to myoclonus and rigidity. Management is largely supportive. Intubation and mechanical ventilation may be necessary for patients with significant altered mental status. Benzodiazepines are indicated for control of agitation and external cooling for sustained hyperthermia. A neuromuscular blocker would be contraindicated since it would preclude the evaluation of seizure activity. Dantrolene and bromocriptine are not recommended in this disorder. Serotonin antagonists such as propranolol, cyproheptadine, and methysergide have been proposed but evidence for benefit is lacking and routine use is not recommended.

### **REFERENCES (9)**

Birmes P, Coppin D, Schmitt L, Lauque D. Serotonin syndrome: a brief review. *CMAJ* 2003;168:1439-1442.

Boyer EW, Shannon M. The serotonin syndrome. *N Engl J Med* 2005;352:1112-1120. Gillman PK. The serotonin syndrome and its treatment. *J Psycopharmacol* 1999;13: 100-109.

RATIONALE (10) Answer: D

Lithium is an alkali metal used in the treatment of bipolar disorder. Peak serum levels are reached in 2 to 4 hours and the drug is excreted in the urine. The greatest risk of lithium ingestion is central nervous system toxicity that can include delirium, tremor, ataxia, increased muscle tone, hyperreflexia, clonus, seizures, and coma. Toxicity is more likely to occur in individuals who chronically ingest lithium than those with a single acute ingestion. Any level ≥ 2.5-4 mmol/L should be considered potentially life-threatening. In this patient with an elevated level and evidence of severe central nervous system toxicity, hemodialysis was appropriately instituted. Although the serum level was decreased effectively by hemodialysis, central nervous system toxicity may not be immediately reversed. This is due to the slow equilibration of intracellular lithium in the brain with the extracellular fluid compartment. At this time in the patient's care, supportive care and observation are the only measures indicated. Repeat lithium levels must be obtained 6-8 hours after dialysis in order to assess for a rebound increase as lithium shifts from the intracellular to extracellular space. If levels increase significantly, further hemodialysis may be warranted. Saline diuresis is not effective in enhancing lithium excretion. Since many patients with lithium toxicity will have dehydration due to the renal effects of lithium (nephrogenic diabetes insipidus), volume replacement is appropriate. Although there is experimental evidence for sodium polystyrene sulphonate to decrease lithium levels, it is not considered part of recommended therapy and may result in significant hypokalemia. Activated charcoal would be useless since lithium is not adsorbed by activated charcoal.

#### **REFERENCES (10)**

Henry GC. Lithium. In *Goldfrank's Toxicologic Emergencies*. 7th ed. Goldfrank LR, Flomenbaum NE, Lewin NA, et al, ed. New York, NY: McGraw-Hill, 2002; 894-900.

Markowitz GS, Radhakrishnan J, Kambham N, et al. Lithium nephrotoxicity: a progressive combined glomerular and tubulointerstitial nephropathy. *J Am Soc Nephrol* 2000; 11:1439-1448.

Scharman EJ. Methods used to decrease lithium absorption or enhance elimination. *J Toxicol Clin Toxicol* 1997;35:601-608.

Timmer RT, Sands JM. Lithium intoxication. J Am Soc Nephrol 1999:10:666-674.

RATIONALE (11)

Answer: E

Increased use of valproic acid for seizure disorders, bipolar disorder, migraine headaches, and neuropathic pain have led to increased reports of overdose. Central nervous system depression is the most common manifestation of toxicity and acute overdose. Higher drug levels are associated with an increased incidence of coma, and intubation and mechanical ventilation may be required. Cerebral edema has been observed in acute overdose and chronic supratherapeutic dosing and may result in death. Central nervous system depression and cerebral edema may be delayed following ingestion as long as 72 hours. Due to the widespread use of sustained-released drug, serial drug levels must be obtained to ensure that levels are declining, even if in the therapeutic range initially. Although hepatotoxicity may be seen with chronic administration, it is rare with acute overdose. Hypernatremia, rather than hyponatremia, has been noted with higher drug levels (levels ≥450 mg/mL).

Additional toxicities that have been described with valproic acid overdoses include hypotension, pancreatitis, metabolic acidosis, and acute renal failure. Management includes close attention to the airway due to the central nervous system depression. Although experience is limited, hemodialysis or combined hemodialysis-hemoperfusion modalities have been reported to be effective in severe toxicity. L-carnitine has been increasingly recommended for the hyperammonemia associated with valproic acid toxicity. Although the exact mechanism is unknown, the ammonemia is generated by an intracellular metabolic defect. There is little evidence that L-carnitine changes the clinical outcome but in severe toxicity it may be considered.

#### **REFERENCES (11)**

Guillaume CPE, Stolk L, DeJere TF, Kooman JP. Successful use of hemodialysis in acute valproic acid intoxication. *J Toxicol Clin Toxicol* 2004;42:335-336.

Szthankrycer MD. Valproic acid toxicity: overview and management. *J Toxicol Clin Toxicol* 2002;40:789-801.

RATIONALE (12)

Answer: C

The appropriate management of acetaminophen overdose is important to prevent hepatic toxicity and mortality. This patient has a potentially toxic ingestion of 15 g, occurring 9 hours prior to presentation. An acetaminophen level should be obtained for comparison with the Rumack-Matthew nomogram to determine potential toxicity. However, the results may not be available rapidly. Because N-acetylcysteine is most effective within 8 hours of ingestion, a loading dose of N-acetylcysteine (oral or IV) should be administered to this patient immediately pending results of the blood level. If the acetaminophen level is in the nontoxic range, no further doses need to be administered.

Liver function tests do not need to be obtained, unless the acetaminophen level is in the toxic range. Liver function tests, if elevated, may also be used to guide therapy in patients who present after 24 hours or who have multiple ingestions over time. In these situations, the Rumack-Matthew nomogram cannot be used to determine toxicity. Activated charcoal can be administered within 4 hours of ingestion and adsorbs acetaminophen. However, it is unlikely to have any significant benefit in this patient who is 9 hours out from ingestion. Flumazenil is an antidote for benzodiazepine toxicity, but it is not needed in this patient who is arousable and has no significant respiratory depression.

#### **REFERENCES (12)**

- Brok J, Buckley N, Gluud C. Interventions for paracetamol (acetaminophen) overdoses. *Cochrane Database Syst Rev* 2002;(3):CD003328.
- Daly FFS, O'Malley GF, Heard K, et al. Prospective evaluation of repeated supratherapeutic acetaminophen (paracetamol) ingestion. *Ann Emerg Med* 2004;44:393.
- Prescott L. Oral or intravenous N-acetylcysteine for acetaminophen poisoning? *Ann Emerg Med* 2005; 45:409-413.

RATIONALE (13)

Answer: D

This patient presents with a typical sympathomimetic syndrome consistent with cocaine or amphetamine intoxication. Clinical findings often include tachycardia, hypertension, hyperthermia, mydriasis, agitation, and psychosis. Other possible complications of cocaine or amphetamine abuse may be difficult to evaluate in the agitated patient. The initial treatment in this patient should include IV or intramuscular lorazepam for sedation. Control of agitation will usually result in a decrease in HR, BP, and temperature. Additional interventions can subsequently be instituted. IV fluids should be administered to establish adequate urine output for possible rhabdomyolysis, an ECG should be obtained to assess for myocardial ischemia, laboratory tests assessing electrolytes, renal function and creatine kinase should be drawn, and a CT scan of the head may be indicated to rule our intracranial injury.

Labetalol may be an appropriate antihypertensive agent in a cocaine or amphetamine abuser who has severe sustained hypertension due to its combined  $\alpha$ - and  $\beta$ -blocking effects. However, hypertension is rarely severe and usually responds to control of agitation. Other  $\beta$ -blockers are not recommended because of the potential concern for worsening of vasoconstriction due to unopposed  $\alpha$ -effects. Haloperidol is not initially indicated for control of agitation in the cocaine abuser. Haloperidol has the potential to lower the seizure threshold and would not be an initial treatment in this patient who has already had a seizure. After agitation is controlled, haloperidol can be considered if the patient manifests psychotic features. Although the patient has experienced a seizure, phenytoin is not indicated. Drug-induced seizures usually do not respond well to phenytoin, and they are usually self-limited. The treatment of choice for drug-induced seizures is benzodiazepines. Although the patient is febrile, acetaminophen would not be considered until agitation is controlled.

#### **REFERENCES (13)**

Chiang WK. Amphetamines. In Goldfrank's Toxicologic Emergencies. 7th ed. Goldfrank LR, Flomenbaum NE, Lewin NA, et al, ed. New York, NY: McGraw-Hill, 2002; 1020-1033.
Hollander JE, Hoffmann RS. Cocaine. In Goldfrank's Toxicologic Emergencies. 7th ed. Goldfrank LR, Flomenbaum NE, Lewin NA, et al, ed. New York, NY: McGraw-Hill, 2002; 1004-1019.
Lange RA, Hillis LD. Cardiovascular complications of cocaine use. N Engl J Med 2001; 345:351-358.
Lineberry TW, Bostwick JM. Methamphetamine abuse: a perfect storm of complications. Mayo Clin Proc 2006;81:77-84.

RATIONALE (14)

Answer: A

Patients presenting with alterations in mental status associated with an ECG that shows widened QRS should be suspected for possible tricyclic antidepressants toxicity. This patient had an overdose of amitriptyline (a tricyclic antidepressant), which is responsible for his neurologic and cardiovascular abnormalities. Most commonly involved drugs include amitriptyline, doxepin, imipramine, and nortriptyline. Tricyclic antidepressants have several actions that can result in neurological changes and in hemodynamic instability. Central nervous systems effects include altered mental status and, in some, advance cases seizures. Cardiovascular effects include ECG changes (sinus tachycardia, white QRS, increased PR, and QT intervals), arrhythmias, and hypotension. Most patients who die from TCA overdoses die secondary to cardiovascular abnormalities. Other clinical findings result from antimuscarinic effects from TCAs and include mydriasis, blurred vision, fever, lethargy, ileus, and urinary retention.

Treatment includes initial stabilization (ABCs), gastrointestinal decontamination activated charcoal), supportive care, and blood alkalinization. Blood alkalinization and TCA toxicity is indicated in patients with wide QRS, in patients with ventricular arrhythmias, or patients with a terminal and replace with R wave in lead aVR>33 mm. Blood alkalinization is usually achieved by administration of sodium bicarbonate as an initial bolus 1-2 mEq/kg and followed by a continuous infusion. The sodium bicarbonate infusion can be tapered once the ECG shows clinical improvement for several hours. bicarbonate can be stopped one the ECG shows clinical improvements or if the pH is between 7.50 to 7.55. Therefore, answer A is correct. Answer B would be incorrect, because there is no evidence of digoxin intoxication. Calcium chloride is usually reserved for patients with calcium channel blocker toxicity. Glucagon may be helpful in patients with β-blockers toxicity, and, finally, naloxone is a reversing agent for benzodiazepine overdose.

#### **REFERENCES (14)**

- Bradberry SM, Thanacoody HK, Watt BE, Thomas S HL, Vale J. Management of the cardiovascular complications of tricyclic antidepressant poisoning: role of sodium biocarbonate. *Toxicol Rev* 2005;24:195-204.
- Greene SL, Dargan PI, Jones AL. Acute poisoning: understanding 90% of cases in a nutshell. *Postgrad Med J* 2005;81:204-216.
- Mills K. Cyclic antidepressants. In: *Critical Care Toxicology*. Brent J, et al, ed. Philadelphia, PA: Mosby, 2005.

# SECTION 12: Pulmonary

# **SECTION 12: PULMONARY**

Instructions: For each question, select the most correct answer.

1. A 62-year-old female presents to the emergency department with chest pain and dyspnea. Based on numerous risk factors for venous thromboembolic disease, including a previous history of pulmonary embolism (PE), an acute PE is diagnosed as the etiology of her symptoms. The patient is also noted to have a fever of 39.1°C(102.4°F).

Which of the following statements about fever in acute PE is most correct?

- A. The patient likely has a pulmonary infarct
- B. It is likely that the patient also has an acute infection
- C. Most patients with acute PE will have a body temperature elevation >100°F
- D. The temperature makes clinical findings supporting deep vein thrombosis more likely to be present
- 2. Which of the following statements about pulmonary embolism (PE) is most correct?
  - A. A clinically significant PE may occur in the absence of right ventricular dilation on echocardiography
  - B. In patients with no prior cardiopulmonary disease, a normal arterial blood gas can exclude PE
  - C. An elevated enzyme-linked immunosorbent assay (ELISA) D-dimer is a useful test in making the diagnosis of acute PE
  - D. A sudden fall in end-tidal carbon dioxide (etCO<sub>2</sub>) may suggest acute PE

- 3. Which one of the following parameters for a patient with acute pulmonary embolism should have the most impact on the decision of whether or not to use thrombolytic therapy (see Figure 1 below)?
  - A. Prior cardiopulmonary disease
  - B. Presence or absence of right ventricular dysfunction on ECG
  - C. The characteristics of the clot shown in the CT scan below
  - D. Presence or absence of arterial hypotension

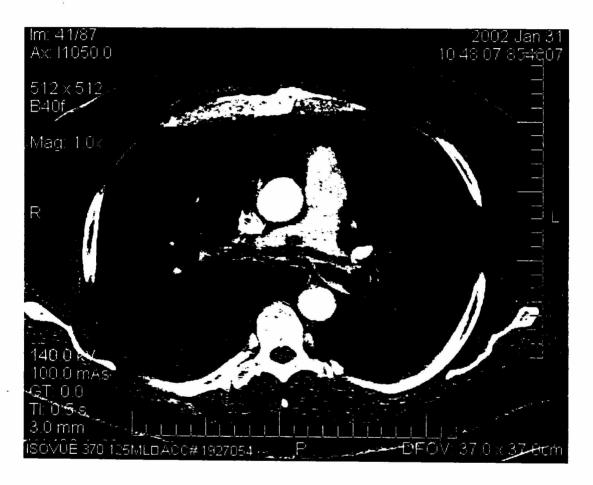


Figure 1.

- 4. Which of the following statements about venous thromboembolic disease in the critically ill patient is most correct?
  - A. If patients with prolonged mechanical ventilation have received appropriate pharmacologic prophylaxis against deep venous thrombosis, deep venous thrombosis (DVT) is uncommon
  - B. A patient with a known DVT and no cardiorespiratory symptoms whatsoever would be unlikely to have abnormal perfusion lung scans
  - C. The sensitivity of Doppler ultrasound in detecting DVT is greater in the presence of signs or symptoms of DVT
  - D. DVT prophylaxis is only necessary in ICU patients receiving mechanical ventilation for longer than 48 hours
- 5. Which one of the following patients is the best candidate for use of noninvasive ventilation?
  - A. 55-year-old male with ST-elevation myocardial infarction with pulmonary edema; BP 90/64 mm Hg, HR 110/min, RR 25/min, pulse oximetry 90% while breathing with a nonrebreather mask; arterial blood gas pH 7.28, Paco<sub>2</sub> 45 mm Hg, and PaO<sub>2</sub> 58 mm Hg
  - B. 68-year-old male receiving home oxygen with chonic obstructive pulmonary disease, COPD exacerbation with mild lethargy; BP 140/92 mm Hg, HR 110/min, RR 30/min, pulse oximetry 96% while breathing with a nonrebreather mask; arterial blood gas pH 7.20, Paco<sub>2</sub> 70 mm Hg, and PaO<sub>2</sub> 120 mm Hg
  - C. 28-year-old female with an exacerbation of asthma; BP 130/88 mm Hg, HR 118/min, RR 20/min, pulse oximetry 97% on 2 L/min nasal cannula; arterial blood gas pH 7.45, Paco<sub>2</sub> 35 mm Hg, and PaO<sub>2</sub> 120 mm Hg
  - D. 59-year-old male with bronchiectasis with worsening shortness of breath and significant ongoing hemoptysis; BP 118/68 mm Hg, HR 105/min, RR 32/min, pulse oximetry 92% while breathing with a nonrebreather mask; arterial blood gas pH 7.42, Paco<sub>2</sub> 30 mm Hg, and PaO<sub>2</sub> 65 mm Hg
  - E. 62-year-old female with coronary artery bypass surgery who was extubated immediately postoperatively but developed respiratory distress 8 hours later; BP 108/70 mm Hg, HR 72/min, RR 25/min, pulse oximetry 94% while breathing with a nonrebreather mask; arterial blood gas pH 7.37, Paco<sub>2</sub> 34 mm Hg, and PaO<sub>2</sub> 76 mm Hg

6. A 62-year-old female is admitted to the ICU with an acute chronic obstructive pulmonary disease exacerbation. An arterial blood gas of 7.15/78/68/96% is obtained during administration of supplemental oxygen. Noninvasive ventilation (expiratory pressure 4 cm H<sub>2</sub>O, and inspiratory pressure 10 cm H<sub>2</sub>O, Fio<sub>2</sub> 50%) is initiated.

An hour later, the nurse notifies you that the patient's BP has dropped from 140/85 to 110/60 mm Hg. On examination, the patient seems more lethargic. The SaO<sub>2</sub> is now 89%. An ECG is obtained and shows T-wave inversions in the anterior leads.

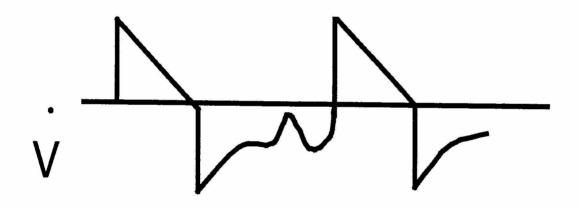
Which of the following is the best next step in this patient's management?

- A. Measure arterial blood gases
- B. Increase oxygen to maintain saturation at 92% or higher
- C. Orally intubate the patient, and provide mechanical ventilator support
- D. Increase the expiratory pressure setting by 5 cm H<sub>2</sub>O
- E. Increase the inspiratory pressure setting by 3 cm H<sub>2</sub>O
- 7. Which of the following is utilized in the calculation of the appropriate tidal volume for a patient with acute respiratory distress syndrome (ARDS) in accordance to ARDSNet recommendations?
  - A. Patient's body mass index
  - B. Patient's weight
  - C. Patient's height
  - D. Patient's age

8. A 64-year-old female, weighing approximately 60 kg, is admitted to the ICU from the emergency department following intubation and initiation of mechanical ventilation. No past medical history is available. Chest radiograph reveals a right lower lobe infiltrate compatible with pneumonia. Ventilator settings are Fio<sub>2</sub> 0.5, assist control volume ventilation, rate 16/min (total assisted rate = 20), tidal volume 600 mL, square inspiratory waveform, peak inspiratory flow rate 80 L/min, and positive end expiratory pressure 5 cm H<sub>2</sub>O. The patient is exhibiting dysychronous breathing. Pulse oximetry oxyhemoglobin saturation is 98%. Flow over time waveform is shown (see figure below).

Which one of the following is most likely to be effective in improving the dysynchronous breathing?

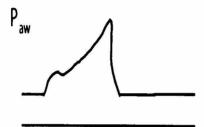
- A. Decreasing peak inspiratory flow rate
- B. Switching to decelerating inspiratory flow waveform
- C. Increasing set ventilator rate to ≥20
- D. Positioning patient with left side down
- E. Increasing set positive end expiratory pressure



9. An 28-year-old male, weighing 80 kg, is admitted to the ICU following intubation and initiation of mechanical ventilation for community-acquired pneumonia associated with severe hypoxemia. His breathing pattern is dysynchronous. Ventilation settings on assist control volume cycled ventilation are F10<sub>2</sub> 0.6, rate 14 (total rate assisted is 25), tidal volume 700 mL, positive end expiratory pressure (PEEP) 10 cm H<sub>2</sub>O, peak inspiratory flow rate 60 L/min, with a square inspiratory flow waveform. Pulse oximetry reveals an oxyhemoglobin saturation of 96%. Pressure over time waveform is shown below.

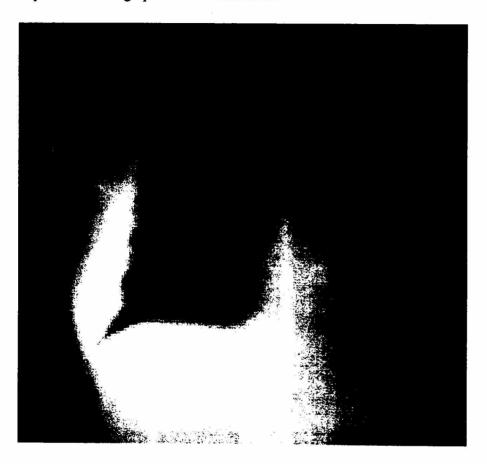
Which one of the following is most likely to relieve the patient's dysynchronous breathing?

- A. Increasing peak inspiratory flow rate (PIFR)
- B. Switching to a decelerating inspiratory flow waveform
- C. Increasing ventilator rate
- D. Increasing PEEP
- E. Decreasing PEEP



- 10. Which one of the following is most correct concerning permissive hypercapnia?
  - A. Permissive hypercapnia is contraindicated in patients with primary metabolic alkalosis
  - B. Permissive hypercapnia should be limited to Paco<sub>2</sub> ≤55 mm Hg in patients with increased intracranial pressure
  - C. Permissive hypercapnia typically becomes less problematic to maintain over time
  - D. Permissive hypercapnia has no effect on PaO,

11. A 20-year-old female is admitted to the ICU after sustaining a closed-head injury in a motor vehicle collision. The patient was intubated in the emergency department (tube depth = 24 cm at upper incisor). Postintubation, chest radiograph is shown in the figure below. Initial emergency department radiograph was unremarkable.



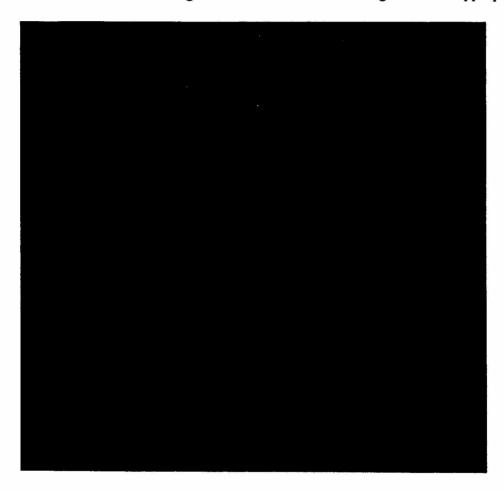
Based on this chest radiograph, which one of the following is most correct?

- A. A thoracostomy tube should be placed on the left side
- B. A thoracostomy tube should be placed on the right side
- C. Emergent bronchoscopy will likely be required
- D. A thoracentesis of the left side should be performed

12. A 57-year-old male with abdominal pain and a diagnosis of acute pancreatitis is admitted to a general medical floor. Despite aggressive fluid resuscitation, the patient developed hypotension, with increased shortness of breath and increasing oxygen requirements. The patient was transferred to the ICU where he was intubated for impending respiratory failure. A chest radiograph obtained after intubation is shown below. Pulmonary artery catheter insertion revealed a pulmonary capillary pulmonary artery occlusion pressure of 6 cm H<sub>2</sub>O. BP improved with further fluid resuscitation and dopamine.

However, the patient developed increased oxygen requirements, with elevated peak and plateau pressures. The patient's actual weight is 80 kg; his ideal body weight is 70 kg.

Which one of the following mechanical ventilation strategies is most appropriate in this patient?



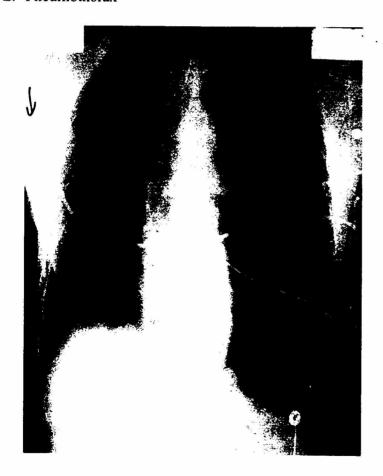
- A. Assist-controlled ventilation, with tidal volumes of 960 mL, and a RR to keep pH around 7.4
- B. Pressure-controlled ventilation, with a maximum combined extrinsic positive end expiratory pressure and pressure control of 35 cm H<sub>2</sub>O, and inverse ratio ventilation of 2:1 to 3:1
- C. Assist-controlled ventilation, with tidal volumes of 420 mL, and bicarbonate therapy if necessary to maintain pH >7.2
- D. Assist-controlled ventilation, with tidal volumes of 840 mL, and intermittent prone positioning

13. A patient is admitted to the ICU after endotracheal intubation for acute chronic obstructive pulmonary disease exacerbation. After 2 days, he is symptomatically better, and weaning from mechanical ventilation is started. Also, glucocorticoid dose is reduced by 50%.

Thirty minutes after changing from assist control to synchronized intermittent mandatory ventilation mode with pressure support, the patient became acutely short of breath. Peak airway pressure increased, and SpO<sub>2</sub> decreased from 95 to 87%. The patient's Fio<sub>2</sub> is increased to 1.0, and a portable supine chest radiograph is done (see the figure below).

Which one of the following is the most likely cause of his acute shortness of breath?

- A. Change from assist control to intermittent mandatory ventilation mode
- B. Exacerbation of chronic obstructive pulmonary disease
- C. Aspiration
- D. Mucus plug
- E. Pneumothorax

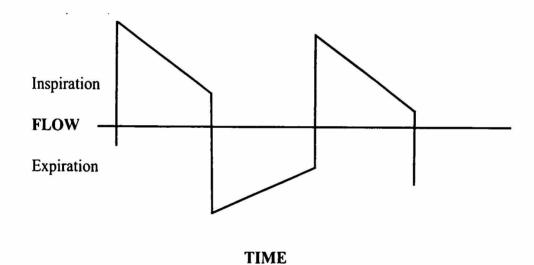


14. A 25-year-old asthmatic woman undergoes emergent laparotomy for a perforated appendix. Postoperatively, she develops profound dyspnea and acute respiratory failure. She is intubated with a rapid sequence induction. Mechanical ventilation was initiated in an assist-control mode (AC 16, TV 550, positive end expiratory pressure 0, Fio, 1.0).

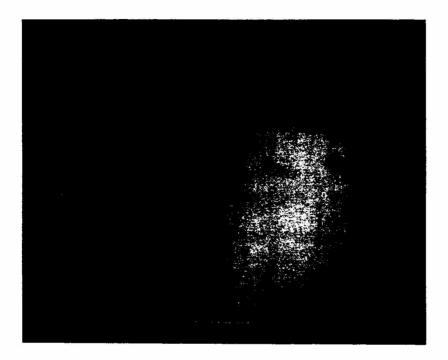
You evaluate the patient in the postanesthesia recovery room. She is deeply sedated (RR 16/min with a set rate of 16/min with the ventilator). You see the following flow graphic waveform displayed on the ventilator (see figure below).

Which one of the following would be most effective in correcting this problem?

- A. Decrease the inspiratory flow rate
- B. Increase tidal volume
- C. Decrease respiratory rate
- D. Increase positive end-expiratory pressure



15. A 56-year-old patient is being weaned from mechanical ventilation. The patient's arterial saturation suddenly drops, requiring high flow oxygen. Breath sounds are diminished over the left chest. A chest radiograph is obtained (below). The lung fields appeared clear on a chest radiograph performed yesterday.



Which of the following is most likely to improve oxygenation?

- A. Needle decompression of the left chest
- B. Tube thoracostomy of the left chest
- C. Position the patient in the left lateral decubitus position
- D. Aggressive mechanical ventilation and suctioning with instillation of normal saline solution

16. A 58-year-old patient is intubated with an exacerbation of his chronic obstructive pulmonary disorder. There is no pneumonia present, but secretions are thick and yellow. Bronchodilator and steroid therapies are initiated. On day 4 of mechanical ventilation, the patient suffers a respiratory distress followed by cardiac arrest. Respiratory support data over the 5 days leading up to the arrest are provided on the following page.

	Day 1	Day 2	Day 3	Day 4
Fio,	0.4	0.4	0.4	0.5
PEEP, cm H <sub>2</sub> O	5	5 .	5	5
O, Saturation	92-95	91-96	90-94	91-95
Peak Inspiratory Pressure, cm H <sub>2</sub> O	22-25	25-28	30-35	42-45
Inspiratory Pplat, cm H <sub>2</sub> O	15-18	15-19	16-18	15-18
RR, breaths/min	18-20	20-24	22-26	24-32

Which one of the following is the most likely reason for the respiratory arrest?

- A. Flash pulmonary edema
- B. Endotracheal tube obstruction
- C. Tension pneumothorax
- D. Pulmonary embolus

17-18. A 27-year-old male presents to the emergency department (brought by family) with complaints of severe weakness. He had a viral illness 1 week ago. Physical examination reveals muscle weakness in both upper and lower extremities. Patient's respirations are rapid and shallow. BP is 150/100 mm Hg; HR 135/min. Arterial blood gases on high flow oxygen reveal a pH of 7.18; PAco<sub>2</sub> of 50 mm Hg; and a PaO<sub>2</sub> of 150 mm Hg.

His electrolyte levels are outlined below:

Sodium: 140 mEq/L Chloride: 118 mEq/L Bicarbonate: 17 mEq/L Potassium: 1.6 mEq/L

The patient's mental status is normal, and he appears to be in mild-to-moderate respiratory distress

The patient is admitted to the ICU to be intubated and mechanically ventilated.

- 17. Which one of the following is the most likely reason for the patient's ventilatory failure?
  - A. Hypokalemia induced by renal tubular acidosis
  - B. Hypokalemic periodic paralysis
  - C. Guillain-Barre syndrome
  - D. Contraction alkalosis associated hypokalemia
- 18. In this patient, which one of the following is most important for preventing iatrogenic complications?
  - A. Choice of potassium formulation for correction of hypokalemia
  - B. Choice of maintenance IV fluids
  - C. Decision as to nutritional supplementation
  - D. Choice of initial mechanical ventilation settings
- 19. In her 34th week of pregnancy, a 41-year-old woman with a history of severe asthma is admitted to the ICU. Since presentation to the emergency department, she has had continued respiratory distress. Nebulized albuterol, ipratropium, and IV corticosteroids were initiated in the emergency department at currently recommended doses. It is now 3 hours since she arrived in the emergency department. Her RR is 34/min, and she has moderate respiratory distress. Her oxygen saturation rate is 92%, with a 100% oxygen nonrebreathing mask.

Which one of the following is most correct concerning the use of heliox?

- A. Heliox is contraindicated in this patient because of the degree of hypoxemia
- B. Heliox will have no effect on PAco,
- C. Heliox has been demonstrated to reduce the incidence of tracheal intubation in randomized trials
- D. Heliox is contraindicated in this patient because of pregnancy

- 20. Which one of following statements is most correct concerning nonpulmonary causes of respiratory failure?
  - A. In patients with abnormalities of the chest wall as the reason for respiratory failure, the diffusing capacity referenced to alveolar volume is significantly decreased
  - B. A quadriplegic patient with a high spinal cord lesion above the origin of the phrenic nerves (C3 to C5) is an ideal candidate for electrophrenic pacing for diaphragm failure
  - C. Respiratory myopathy is not a physiologic process contributing to respiratory failure in severe hypothyroidism
  - D. Respiratory alternans (use of the diaphragm for some breaths and not for others) is characterized by a tidal volume variation of at least 500 mL
- 21. A 55-year-old female is admitted to the ICU from the emergency department with severe acute respiratory distress syndrome. She is currently being mechanically ventilated with a tidal volume of 6 mL/kg predicted body weight and a positive end expiratory pressure of 14 cm H<sub>2</sub>O, with a Fio<sub>2</sub> of 0.6. Her inspiratory plateau pressure is 32 cm H<sub>2</sub>O.

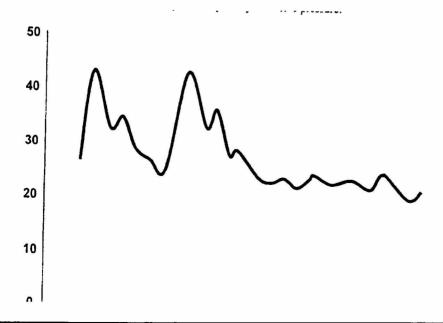
Which one of the following is most likely to improve survival in this patient?

- A. Inserting a central venous catheter and target a central venous pressure of <4 mm Hg as long as the patient remains hemodynamically stable with good tissue perfusion
- B. Inhaled nitric oxide .
- C. Prone positioning
- D. Decreasing tidal volume

- 22. Which one of the following statements is most correct concerning the most appropriate clinical strategy for ventilator-associated pneumonia?
  - A. A reliable tracheal aspirate Gram stain cannot be used to direct initial empiric antimicrobial therapy
  - B. Negative tracheal aspirate culture data in a patient without a recent change in antibiotics offers minimal utility in ruling out ventilator-associated pneumonia
  - C. Regardless of the pretest probability of pneumonia, the absence of bacteria on a microscopic examination of the lower respiratory tract (bronchoalveolar lavage) samples allows withholding antibiotic therapy
  - D. Tracheal aspirate culture data almost always contain the pathogens found in quantitative invasive lower respiratory tract cultures
- 23. A 56-year-old patient with long-standing chronic obstructive pulmonary disorder is intubated for respiratory failure due to community-acquired pneumonia. Chest radiograph reveals a dense right lower lobe infiltrate and prominent pulmonary arteries. Patient is severely hypoxemic and is receiving F10<sub>2</sub> of 0.8 to maintain oxygenation. Urine output remains low, despite repeated fluid bolus. Patient is heavily sedated without evidence of significant auto-positive end expiratory pressure. A pulmonary catheter is inserted, and the waveform is shown in the figure below.

Which one of the following is most appropriate treatment at this time?

- A. Administer IV furosemide
- B. Obtain an echocardiogram to rule out pericardial tamponade
- C. Continue fluid bolus
- D. Repeat measurement of pulmonary artery occlusive pressure

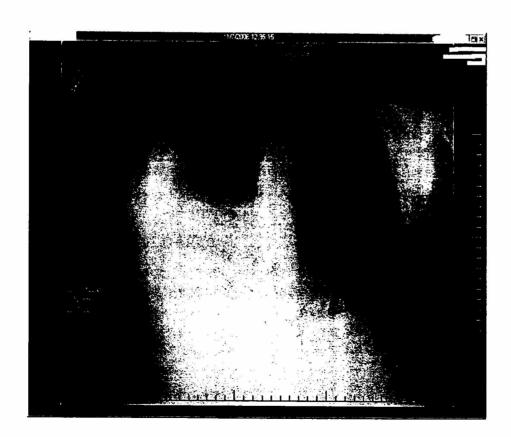


- 24. A 70-year-old female is postoperative day 2 following a carotid endarterectomy with reexploration required on day 1. Rapidly progressive hypoxemia develops. She is intubated and mechanical ventilation is initiated. SaO<sub>2</sub> is 82% on 100% oxygen. She has decreased breath sounds on the right, with crackles (chest radiograph obtained prior to intubation is shown in the figure below). Mechanical ventilation has been increased. Current ventilator settings are as follows:
  - Volume assist control, 6.0 mL/kg ideal body weight;
  - Positive end expiratory pressure, 14 cm H<sub>2</sub>O;
  - Set rate 20 with actual rate 24;
  - F10, 1.0; I:E ratio 1:2; and
  - Peak inspiratory pressure = 32 cm H<sub>2</sub>O.

ABGs reveal: pH 7.32, Paco, 49 mm Hg, Pao, 50 mm Hg.

Which one of the following is least appropriate at this time?

- A. Put right side down
- B. Prone position
- C. Recruitment maneuver
- D. Increase positive end expiratory pressure



- 25. Which one of the following is most correct concerning mechanical ventilation in patients with acute respiratory distress syndrome (ARDS)?
  - A. Prone positioning produces optimal clinical effect with daily use of 6-10 hours
  - B. A recruitment maneuver (sustained elevated end inspiratory pressure) with no change in positive end expiratory pressure (PEEP) produces a short lived improvement in oxygenation
  - C. Compared with the PEEP settings used in the first ARDSnet clinical trial (compared low versus conventional test volume), higher PEEP settings offer no additional improvement in Pao.
  - D. There is no evidence that inspiratory plateau pressure correlates with outcome in ARDS
- 26. Which one of the following is not associated with an increased risk of developing hospital-acquired pneumonia?
  - A. Reintubation
  - B. Noninvasive ventilation
  - C. Subglottic secretion pooling
  - D. Endotracheal tube cuff pressure <20 cm H<sub>2</sub>O
  - E. Recumbent position

# **SECTION 12: PULMONARY**

#### **ANSWERS:**

1-D; 2-D; 3-D; 4-C; 5-B; 6-C; 7-C; 8-E; 9-A; 10-C; 11-C; 12-C; 13-E; 14-C; 15-D; 16-B; 17-A; 18-D; 19-A; 20-B; 21-D; 22-D; 23-D; 24-A; 25-B; 26-B

RATIONALE (1)

Answer: D

Fever can be observed in acute pulmonary embolism (PE), but a high fever is uncommon. In a study of 312 patients with acute PE documented via angiography who had no infection, 86% were noted to have body temperatures <37.8°C (100.0°F); 8% had body temperatures from 37.8 to 38.3°C (100.0 to 100.9°F). Only 4 of 312 patients had body temperatures  $\geq$ 38.9°C (102.0°F), and one had a temperature of  $\geq$ 39.4°C (103.0°F). Symptoms of deep vein thrombosis were more likely in subjects with temperature elevations. Pulmonary infarction was not more common.

Although the observation of fever > 38.9°C (102°F) does not necessarily impact the likelihood of acute PE, PE is the an uncommon cause of fever this high. Therefore, a comprehensive diagnostic evaluation needs to be considered in order to exclude the possibility of a bacterial or other infection. The study cited noted that subjects were more likely to have a significant fever with acute PE, if they also had a concurrent symptomatic deep venous thrombosis.

#### REFERENCE (1)

Stein PD, Afzal A, Henry JW, Villareal CG. Fever in acute pulmonary embolism. Chest 2000;117:39-42.

RATIONALE (2)

Answer: D

As obstructive shock is the mechanism by which the vast majority of fatal cases of acute pulmonary embolism (PE) cause death, the mortality risk in acute PE is a function of the degree of obstructive shock that can be evident in acute right ventricular dilation. While the absence of right ventricular dilation virtually excludes acute PE as an etiology of shock, a clinically significant (i.e. symptomatic) PE could still occur in the absence of right ventricular dilation. This is particularly true in the case of a patient with no prior cardiopulmonary disease and a good cardiopulmonary reserve.

Arterial blood gas analysis does not provide adequate discriminant value to exclude acute PE, particularly in patients with no prior cardiopulmonary disease. In a subgroup analysis of the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) data, 38% of subjects with no prior cardiopulmonary disease and normal blood gases were found to have PE documented via angiography. Although the finding of a normal blood gas with PE was less likely in patients with prior cardiopulmonary disease, an arterial blood gas analysis was still found to be an inadequate test to exclude PE.

In the emergency department setting, a negative ELISA D-dimer in a patient with low clinical suspicion of PE may be useful, as it is the negative predictive value of this test which is clinically meaningful. However, an elevated ELISA D-dimer is minimally useful, as the positive predictive value is not nearly as good. This is due to the multitude of other etiologies for D-dimer elevation, including any cause of systemic inflammation.

An acute decrease in end-tidal CO<sub>2</sub> (etCO<sub>2</sub>) may suggest acute PE. It may decrease with a massive acute PE. This is because gas exchange will be occurring in areas of nonperfused lung without picking up much carbon dioxide. The etCO<sub>2</sub> has been found to return to normal range with successful thrombolysis.

## **REFERENCES (2)**

- Oger E, Leroyer C, Bressollette L, et al. Evaluation of a new, rapid, and quantative D-dimer tests in patients with suspected pulmonary embolism. *Am J Respir Crit Care Med* 1998;158:65-70.

  Stein PD, Goldhaber SZ, Henry JW, Arterial blood gas analysis in the accessory of the second state.
- Stein PD, Goldhaber SZ, Henry JW. Arterial blood gas analysis in the assessment of suspected acute pulmonary embolism. *Chest* 1996;109:78-81.
- Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. *Chest* 2002;121:877-905.

RATIONALE (3) Answer: D

The presence of shock is the most well-accepted trigger for thrombolytic therapy administration in the setting of an acute pulmonary embolism (PE). The mechanism by which a patient with acute PE experiences death is mediated by obstructive shock, where the acute right ventricular obstruction prevents the filling of the left heart and decreases cardiac output, resulting in death.

Death by refractory hypoxemia is uncommon. The presence of arterial hypotension is a clear-cut indicator for initiating thrombolysis for acute PE (in the absence of absolute contraindications). The presence of right ventricular dysfunction does impact the decision to use thrombolysis, because a normal-appearing right ventricle would virtually exclude PE as the etiology of shock.

Whether or not right ventricular dysfunction alone should be a criteria to initiate thrombolytic therapy is extremely controversial. As effective anticoagulation is likely to abate recurrent thrombolytic events, and right ventricular dysfunction is quite common in acute PE of various severities, it is likely that, because of a 2-3% risk of intracranial hemorrhage, reserving thrombolytic therapy for a patient with hypotension is a prudent decision. Nonetheless, this remains an area of ongoing investigation.

The presence of cardiopulmonary disease contributes to the mortality risk from acute PE, as those with prior cardiopulmonary disease and much less cardiopulmonary reserve are more likely to have hemodynamically significant PE with a much smaller clot burden. The central location of the PE on a CT scan (i.e. "saddle" embolus—as shown in the figure) is not by itself a factor that would require the use of thrombolytic therapy. Rather, the apparently large size or central location of the PE on CT scan could prompt an ECG that could risk-stratify the patient.

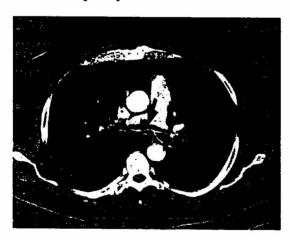


Figure 1. A CT scan of the chest demonstrating acute central pulmonary embolism (arrows).

#### **REFERENCES (3)**

Segal JB, Streiff MB, Hoffman LV, et al. Management of venous thromboembolism: A systematic review for a practice guideline. *Ann Intern Med* 2007;146:211-222.

Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. *Chest* 2002;121:877-905.

Wood KE. The presence of shock defines the threshold to initiate thrombolytic therapy in patients with pulmonary embolism. *Intensive Care Med* 2002;28:1547-1551.

RATIONALE (4)

Answer: C

Venous thromboembolic disease is a spectrum, ranging from deep venous thrombosis (DVT) to acute pulmonary embolism. Patients with DVT are likely to be having subclinical pulmonary embolism (PE), even in the absence of cardiorespiratory symptoms. This has been documented in prospective studies involving perfusion lung scans. Therefore, DVT and PE should not be thought of as mutually exclusive events.

DVT is common in critically ill patients. Surveillance studies of ICU populations have identified clinically silent DVT, even with what appears to be adequate prophylactic therapy. In particular, it has been demonstrated that patients with prolonged mechanical ventilation have a high incidence of DVT, even with appropriate pharmacologic prophylaxis therapies. Clinicians should therefore have a high index of suspicion for silent DVT in these patients.

The sensitivity of Doppler ultrasound to detect DVT depends on the clinical scenario. The sensitivity of Doppler ultrasound is much higher in patients with signs or symptoms of DVT. The sensitivity decreases somewhat in subjects with no signs or symptoms of DVT. Doppler ultrasound is still considered a first-line test regardless of clinical findings.

Prospective studies investigating rates of DVT development in hospitalized patients have found much higher rates of DVT with the use of venography as compared to Doppler ultrasound, probably because of the lower sensitivity in patients without symptoms. Prophylaxis against DVT should be given to all patients in the ICU. Patients with a contraindication to pharmacologic prophylaxis should have leg compression devices. This does not just pertain to those with prolonged mechanical ventilation.

## REFERENCES (4)

- Cook D, Crowther M, Meade M, et al. Deep venous thrombosis in medical-surgical critically ill patients: prevalence, incidence, and risk factors. *Crit Care Med* 2005;33:1565-1571.
- Harris B, Bailey D, Miles S, et al. Objective analysis of tomographic ventilation-perfusion scintigraphy in pulmonary embolism. *Am J Respir Crit Care Med* 2007;175:1173-1180.
- Ibrahim EH, Iregui M, Prentice D, et al. Deep vein thrombosis during prolonged mechanical ventilation despite prophylaxis. Crit Care Med 2002;30:771-774.
- Meignan M, Rosso J, Gauthier A, et al. Systematic lung scan reveal a high frequency of silent pulmonary embolism in patients with proximal deep venous thrombosis. *Arch Intern Med* 2000;160:159-164.
- Proceedings of the seventh ACCP conference on antithrombotic and thrombolytic therapy: evidence-based guidelines. *Chest* 2004;126:172S-696S.

RATIONALE (5)

Answer: B

It is important to know the indications as well as the contraindications for noninvasive positive pressure ventilation (NPPV), and the respiratory conditions in which NPPV offers benefit. Patients with chonic obstructive pulmonary disease (COPD) exacerbations respond well to NPPV, with decreased rates of intubation and improved mortality. Although depressed mental status and inability to cooperate are typical contraindications for NPPV, the patient described has only mild lethargy, which may be related to the increased CO<sub>2</sub>, partly from over oxygenation-induced increase in dead space.

Recent studies also suggest that NPPV can be utilized effectively in selected patients with a depressed level of consciousness. Other contraindications to NPPV are hemodynamic instability, as illustrated by the 55-year-old man with ST-elevation myocardial infarction with pulmonary edema. This patient is in cardiogenic shock with a metabolic acidosis, and should be immediately intubated.

The inability to control secretions, vomiting, and hemoptysis preclude use of NPPV because of aspiration risk. NPPV is unlikely to benefit the patient with mild disease, as in the asthmatic patient presented. In asthma, NPPV should be reserved for patients with moderate to severe asthma who fail to respond to initial therapy and who do not qualify for immediate intubation.

Several studies have shown that NPPV does not prevent reintubation in patients with postextubation respiratory failure.

#### **REFERENCES (5)**

- Caples SM, Gay PC. Noninvasive ventilation in the intensive care unit: a concise review. *Crit Care Med* 2005;33:2651-2658.
- Diaz GG, Alcaraz AC, Talavera JCP, et al. Noninvasive positive-pressure ventilation to treat hypercapnic coma secondary to respiratory failure. *Chest* 2005;127:952-960.
- Estaban A, Frutos-Vivar F, Ferguson N, et al. Noninvasive positive-pressure ventilation for respiratory failure after extubation. *N Engl J Med* 2004;350:2452-2460.
- Keenan S, Powers C, McCormack DG, Block G. Noninvasive positive-pressure ventilation for postextubation respiratory distress. *JAMA* 2002;287:3238-3244.
- Liesching T, Kwok H, Hill NS. Acute applications of noninvasive positive pressure ventilation. *Chest* 2003;124:699-713.
- Scala R, Naldi M, Archinucci I, et al. Noninvasive positive pressure ventilation in patients with acute exacerbations of COPD and varying levels of consciousness. *Chest* 2005;128:1657-1666.

RATIONALE (6)

Answer: C

The patient in this question was initially placed on noninvasive positive pressure ventilation (NPPV) for treatment of an acute chronic obstructive pulmonary disease (COPD) exacerbation. After initiation with appropriate settings, the patient develops hemodynamic changes that include a drop in blood pressure, ECG changes suggestive of ischemia, and a worsening clinical status as evidenced by increased lethargy and increased hypoxemia. Although the institution of NPPV is often beneficial for patients with COPD exacerbations, it is important to recognize when to proceed with endotracheal intubation and provision of conventional mechanical ventilator support. Studies have shown that delaying needed intubation can add to patient morbidity and mortality.

Contraindications and factors that may indicate a high risk of failure with NPPV include the following: respiratory arrest, severe acid-base abnormalities, medically unstable patient (hemodynamic instability, uncontrolled gastrointestinal bleed, acute myocardial ischemia, uncontrolled arrhythmias), inability to protect airway, excessive secretions, uncooperative or agitated patient, and recent upper airway or upper gastrointestinal surgery.

This patient is showing clinical deterioration, with evidence of hemodynamic instability and myocardial ischemia. Therefore, he is not a candidate to continue on NPPV and should be intubated immediately. In addition, predictors of failures of NPPV in patients with acute exacerbation of COPD include a respiration rate >35/min, an APACHE II score >29, and Glasgow coma scale score <11.

#### REFERENCES (6)

- British Thoracic Society Standards of Care Committee. Non-invasive ventilation in acute respiratory failure. *Thorax* 2002;57:192-211.
- Confalonieri M, Garuti G, Cattaruzza MS, et al. A chart of failure risk for noninvasive ventilation in patients with COPD exacerbation. *Eur Respir J* 2005;25:348-355.
- Keenan SP, Sinuff T, Cook DJ, Hill NS. Which patients with acute exacerbation of chronic obstructive pulmonary disease benefit from noninvasive positive-pressure ventilation? A systematic review of the literature. *Ann Intern Med* 2003;138:861-870.
- Lightowler JV, Wedzicha JA, Elliott MW, Ram FS. Non-invasive positive pressure ventilation to treat respiratory failure resulting from exacerbations of chronic obstructive pulmonary disease: Cochrane systematic review and metaanalysis. *BMJ*.2003;326:185.

RATIONALE (7)

Answer: C

The Acute Respiratory Distress Syndrome (ARDS) Network clinical trial of low tidal volumes demonstrated significant improvements in mortality when low volume ventilation was utilized in patients with ARDS. In that study patients were given a tidal volume of 6 mL/kg of predicted body weight (PBW). The formulas for PBW are given below:

- Male = 50 + 2.3 [height (inches) -60] or 50 + 0.91 [height (cm) -152.4]
- Female = 45.5 + 2.3 [height (inches) -60] or 45.5 = 0.91 [height (cm) -152.4]

It is important to utilize the PBW when calculating tidal volume for patients with ARDS, because patients who are volume overloaded or who have a high body mass index may have a much higher actual body weight than a PBW. In those cases, the use of an actual weight may provide deleterious tidal volumes to the patient. In order to calculate the PBW, one must know the patient's height.

#### **REFERENCES (7)**

Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 2002;342:1301-1308.

Kam E, Eslick G, James A, Benson J. Acute respiratory distress syndrome (ARDS) and low tidal volume ventilation: the debate about weight. *Intensive Care Med* 2004;30:1502.

RATIONALE (8)

Answer: E

This flow over time waveform demonstrates the presence of auto positive end expiratory pressure (auto-PEEP), signified by significant expiratory flow still occurring at end expiration (see figure below, arrow 2). In addition, it demonstrates a dramatic drop in flow rate that occurs near the end of expiration which is not associated with inspiratory triggering (see figure below, arrow 1). This occurs because the patient has made a significant inspiratory effort, creating a negative intrapleural pressure, and therefore decelerating the expiratory flow rate. The patient's effort, however, does not trigger the next inspiration and subsequently subsides, followed by an unassisted ventilator triggering based on time interval from last inspiration.

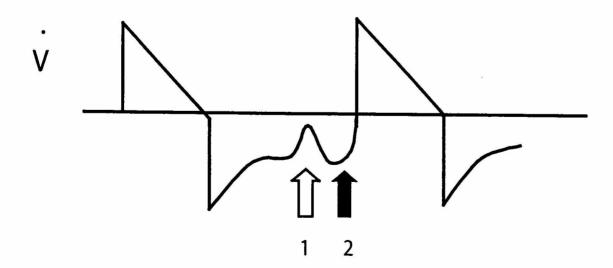
Since the patient must first overcome the auto-PEEP before encountering the normal triggering mechanisms, the patient's work of breathing may be offset to some degree by increasing the set ventilator PEEP, because the degree of inspiration effort to trigger is decreased as the difference between set PEEP and total PEEP decreases. This occurs because the ventilator now triggers from the set PEEP level (plus some change in pressure or flow) instead of from atmospheric pressure.

Decreasing the peak inspiratory flow rate in a patient on volume ventilation would have no effect on this cause of dysynchrony, but in fact would worsen the auto-PEEP by prolonging inspiration.

The inspiratory flow waveform is already set as decelerating flow. Switching to a square inspiratory flow waveform would, however, improve auto-PEEP, as it would shorten the inspiratory time.

Increasing the set ventilator rate to 20 would also have no effect in this patient, as she is already breathing at a rate of 20. If the rate was increased higher than 20, it would exacerbate the auto-PEEP by further decreasing expiratory time.

Positioning the patient left side down would have no predictable effect on this clinical problem.



#### **REFERENCES (8)**

- Aslanian P, et al. Effects of flow triggering on breathing effort. Am J Respir Crit Care Med 1998;157:135-143.
- Chao DC, Scheinhorn DJ, Stearn-Hassenpflug M. Patient-ventilator trigger asynchrony in prolonged mechanical ventilation. *Chest* 1997;112:1592-1599.
- Hess DR, Medoff BD. Mechanical ventilation of the patient with chronic obstructive pulmonary disease. Respir Care Clin North Am 1998;4:439-473.
- Valta P, Corbeil C, Lavoie A, et al. Detection of expiratory flow limitation during mechanical ventilation. *Am J Respir Crit Care* 1994;150:1311-1317.

RATIONALE (9)

Answer: A

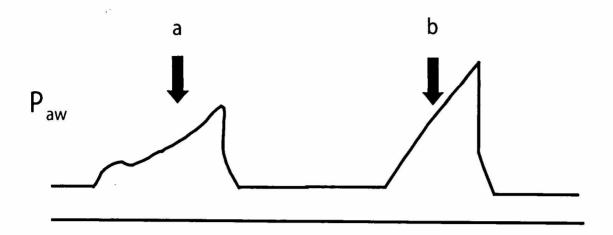
The pressure over time waveform shown in this patient is typical for volume ventilation with rising pressure throughout inspiration. The waveform, however, exhibits a decay in pressure occurring after the onset of inspiration (see figure, arrow a). This drop in pressure is related to significant inspiratory effort occurring after inspiration is triggered. This typically occurs when a patient is not satisfied with the inspiratory flow rate setting.

This is particularly problematic in young healthy individuals with good respiratory muscle strength who are in respiratory distress. Increasing the peak inspiratory flow rate (PIFR) should accommodate the patient's desires and decrease dysynchrony (see figure, arrow b). Note that in addition to smoothing out the inspiratory flow waveform, the peak inspiration pressure also rises with the higher rate. However, tidal volume and inspiratory plateau pressure do not change.

Switching to a decelerating inspiratory flow waveform will likely worsen dyssynchrony, as the PIFR will steadily decrease after onset of inspiration.

Increasing the set ventilator rate, unless it is increased higher then 30 (current assisted rate), would have no effect on any parameters, and if increased above 30 would have no effect on dysnychrony.

Although this patient's positive end expiratory pressure might need to be increased, it is not a reason for the dysynchrony, and with a Fio<sub>2</sub> of 0.6 and oxyhemoglobin saturation of 96%, this is not a critical issue. Decreasing the positive end expiratory pressure to less than 10 cm H<sub>2</sub>O with an Fio<sub>2</sub> of 0.6 is probably not an appropriate intervention.



#### **REFERENCES (9)**

Jubran A. Advances in respiratory monitoring during mechanical ventilation. *Chest* 1999;116:1416-1425.

Tobin MJ, Jubran A, Laghi F. Patient-ventilator interaction. Am J Respir Crit Care Med 2001;163:1059-1063.

Rau JL. Inspiratory flow patterns: The "shape" of ventilation. Respir Care 1993;38:132-140.

RATIONALE (10)

Answer: C

Permissive hypercapnia is a clinical management tool that is sometimes employed to limit tidal volume and inspiratory plateau pressure in severe acute respiratory distress syndrome and severe asthma induced auto-PEEP. Permissive hypercapnia is the process of accepting a higher Paco<sub>2</sub> and a lower pH in order to have a lower tidal volume and lower inspiratory plateau pressure. Because the primary metabolic limitation to permissive hypercapnia is the acidema associated with the rising Paco<sub>2</sub>, employing permissive hypercapnia in patients with normal renal function becomes less problematic over time as the normal kidney responds to the rising Paco<sub>2</sub> by retaining bicarbonate and raising pH. Tidal volume may then be lowered and Paco<sub>2</sub> further raised over time as bicarbonate retention occurs.

Likewise, in patients with preexisting primarily metabolic alkalosis, institution of permissive hypercapnia is facilitated.

Increased intracranial pressure is a contraindication to any degree of permissive hypercapnia, since a rise in Paco<sub>2</sub> will produce cerebral vasodilatation, increased cerebral blood volume, and further increases intracranial pressure.

Because Paco<sub>2</sub> is to some extent dependant upon alveolar ventilation, permissive hypercapnia would be expected to decrease PaO<sub>2</sub> to a small extent.

#### REFERENCES (10)

Balk RA. Permissive hypercapnia: an alternative ventilatory mode for the management of acute lung injury and acute airflow obstruction. *Clin Pulmonary Med* 1997;4:29-33.

Hickling KG. Low volume ventilation with permissive hypercapnia in the adult respiratory distress syndrome. Clin Intensive Care 1992;3:67-78.

Roupie E, Dambrosio M, Servillo G, et al. Titration of tidal volume and induced hypercapnia in acute respiratory distress syndrome. Am J Respir Crit Care Med 1995;152:121-128.

Wiener C. Ventilatory management of respiratory failure in asthma. JAMA 1993;269:2128-2131.

RATIONALE (11)

Answer: C

The patient's endotracheal tube has advanced into the right mainstem bronchus (somewhat difficult to see on figure but inferred by appearance of chest radiograph and ET tube depth). The primary clue to this diagnosis is its occurrence temporally related to intubation. This has caused collapse of the left lung (atelectasis), with a shifting of the mediastinum toward the left. An elevation of the left hemidiaphragm and a narrowing of the left intercostals spaces is also evident on the radiograph. Normal initial ET tube depth in a female is 22 cm at upper incisors.

The indicated intervention is to withdraw the endotracheal tube to a position 2-4 cm above the carina. This will usually re-expand the left lung.

Total opacification of a lung could also be secondary to fluid (or blood) in the pleural space, although in this situation the mediastinum would be shifted to the contralateral side.

Chest tube thoracostomy or thoracentesis are not indicated, and could produce significant complications in this scenario. Bronchoscopy is not necessary, since the problem can be identified on examination of the radiograph.

#### **REFERENCES (11)**

Herman P, Khan A. Critical care radiology. In Dantzker D, Scharf S, eds. *Cardiopulmonary Critical Care*. Philadelphia, PA: WB Saunders; 1998.

Rothenberg D, Barboi C. Airway management and endotracheal intubation. In Parrillo JE, Dellinger RP, eds. *Critical Care Medicine*. St. Louis, MO; 2001:16-35.

RATIONALE (12)

Answer: C

This patient has developed acute respiratory distress syndrome (ARDS), likely related to systemic inflammatory response syndrome and acute pancreatitis. With the advent of mechanical ventilation, ARDS-associated mortality no longer is related to respiratory failure, but to multiple organ failure and sepsis.

Recently, there has been growing concern that ventilation strategies using large tidal volumes can cause increased injury and lead to increased mortality. As lung compliance decreases in ARDS, traditional volume-cycled ventilation employing large tidal volumes (10-15 mL/kg) require elevated positive pressures, which damages alveolar tissue. High peak and plateau pressures (plateau pressures >35 cm H<sub>2</sub>O) cause overdistension of lung tissue, and lead to further damage through the release of inflammatory mediators.

Various ventilator strategies have been used to try to reduce high-pressure injury and volutrauma, in hopes of improving mortality. Previously, newer ventilation techniques, including jet ventilation, pressure control with inverse ratio ventilation, and prone positioning have not shown any improvement in mortality. However, recent data from randomized studies suggest potential benefit of prone positioning in patients with more severe disease. No information is given in this case on severity.

Recent work published by the Acute Respiratory Distress Syndrome Network of the National Heart, Lung and Blood Institute (ARDSNet) showed that a volume-restricted ventilation strategy reduced mortality. In their study, involving 10 university centers, patients were randomly assigned to either traditional volume ventilation strategy (12 mL/kg of ideal body weight, maintaining plateau pressures <50 cm  $H_2O$ ), or low tidal volume ventilation (6 mL/kg, maintaining plateau pressures <30 cm  $H_2O$ ). The low tidal volume group had a reduced overall mortality (31.0% vs 39.8, p <0.007). Furthermore, the investigators were able to reach the reduced tidal volumes (6.2 ± 0.8 mL) and reduced plateau pressures (25 ± 6) in the study group. Interleukin-6 (IL-6) levels were also reduced, suggesting a decrease in inflammatory mediator production. Permissive hypercapnia was tolerated, although patients were administered bicarbonate therapy to maintain pH >7.2.

## **REFERENCES (12)**

- Amato, MB, Barbas CS, Medeiros DM, et al. Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. N Engl J Med 1998;338:347-354.
- ARDS Network. Ventilation with lower tidal volumes as compared with traditional volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 2000;342:1301-1308.
- Dreyfus D, Soler P, Basset G. High inflation pressure pulmonary edema: respective effects of high airway pressure, high tidal volume, and positive end-expiratory pressure. *Am Rev Respir Dis* 1988;137:1159-1164.
- Gattinoni L, Tognoni G, Pesenti A, et al. Effect of prone positioning on the survival of patients with acute respiratory failure. N Engl J Med 2001;345:568-573.
- Parker JC, Hernandez LA, Longenecker GL, et al. Lung edema caused by high peak inspiratory pressures in dogs: role for increased microvascular filtration pressure and permeability. Am Rev Resp Dis 1990;142:321-328.
- Whitcomb DC. Acute pancreatitis. N Engl J Med 2006;354:2142-50.

RATIONALE (13) Answer: E

The chest radiograph shows a deep sulcus sign in the left base, which on supine radiograph is compatible with the diagnosis of pneumothorax. Because of the position of the free air, portable chest radiograph detection of a pneumothorax in a supine position is difficult. In patients who can sit up and cooperate by holding their breath after exhalation, a pneumothorax can usually be easily detected in the apical location. In the supine patient, Tocino and colleagues found the most common distribution of pneumothoraces in ICU patients to be anteromedial (38%), causing the deep sulcus sign, subpulmonic (26%), and apical location (22%).

Increasing airway pressures, patient ventilator dyssynchrony, and hypoxemia can be seen with all of the potential answers and will not help in making the diagnosis.

No consolidation or infiltrates are seen, making mucus plugs and aspiration unlikely.

An acute exacerbation of chronic obstructive pulmonary disease would not produce a deep sulcus sign.

Since the patient tolerated intermittent mandatory ventilation mode for 30 minutes before becoming short of breath, the abrupt onset of the shortness of breath is probably not due to the change in ventilator mode.

In addition to the presence of a pleural line or the deep sulcus sign, if the chest radiograph shows a unilateral increase in hemithorax volume, a well-defined diaphragm despite lung consolidation, or if 1 lung is more lucent than the other, when compared with a recent old chest radiograph, a pneumothorax should be suspected. If a pneumothorax is suspected but cannot be confirmed by chest radiograph, a CT scan of the chest can help identify the presence of pneumothorax. Cross-table lateral chest radiograph may be useful for patients who are too unstable for a CT scan.

#### **REFERENCES (13)**

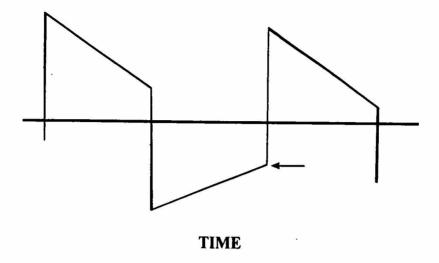
Sandur S, Stoller JK. Pulmonary complications of mechanical ventilation. *Clin Chest Med* 1999;20:223-247.

Strange C. Pleural complications in the intensive care unit. Clin Chest Med 1999;20:317-327. Tocino IM, Miller MH, Fairfax WR. Distribution of pneumothorax in the supine and semirecumbent critically ill adult. AJR 1985;144:901-905.

RATIONALE (14)

Answer: C

The arrow on the flow-time diagram below indicates intrinsic positive end-expiratory pressure (intrinsic positive end-expiratory pressure (PEEP) or auto-PEEP).



Intrinsic PEEP is present when the end-expiratory lung volume represents a volume at which air would normally continue to escape if there was no impediment to expiration. When the expiratory time is insufficient to allow full exhalation of a ventilator breath, expiratory flow is still occurring when the next ventilator breath is delivered (arrow). Since the expiratory flow does not reach zero, there is also a pressure gradient at end-expiration. Therefore, PEEP exists even when it is not set on the ventilator (or in excess of what is set on the ventilator). The presence or absence of intrinsic PEEP may be confirmed by checking graphic flow waveforms on the ventilator.

Since severe bronchospasm increases the necessary expiratory time, patients with either status asthmaticus (as in this case scenario), or severe chronic obstructive pulmonary disease exacerbation are at risk for intrinsic PEEP.

The patient in this case scenario should be ventilated with a strategy to limit intrinsic PEEP, by decreasing inspiratory time relative to expiratory time. This is best accomplished by decreasing the respiratory rate, giving the patient more time to exhale between breaths. Decreasing the tidal volume will also result in a small improvement in intrinsic PEEP, but to a much lesser degree than decreasing the rate.

With volume-cycled ventilation, the inspiratory time is also dependent on the peak inspiratory flow rate, which determines how fast a breath will be delivered. The choice of inspiratory flow waveform also influences inspirator:expiratory ratio. When providing volume-cycled ventilation in the presence of a bronchospasm-induced intrinsic PEEP, the peak inspiratory flow rate should be set at 80-100 L/min, with a square waveform, in order to limit the inspiratory time, leaving more time in the respiratory cycle for expiration.

However, it must be emphasized that the inspirator:expiratory ratio should not be the primary goal in managing intrinsic PEEP. Rather, the chief determinant of intrinsic PEEP is the *absolute* time for expiration of each breath. That is why decreasing the respiratory rate is the critical intervention.

#### **REFERENCES (14)**

Pepe PE, Marini JJ. Occult positive end-expiratory pressure in mechanically ventilated patients with airflow obstruction: the auto-PEEP effect. *Am Rev Respir Dis* 1982;126:166-170.

Stather DR, Stewart TE: Clinical review: Mechanical ventilation in severe asthma. *Crit Care* 2005;9:581-587.

RATIONALE (15)

Answer: D

This patient on mechanical ventilation has a "white-out" of the entire left lung. While in some clinical scenarios it would be possible that this radiographic finding could represent a massive amount of fluid in the pleural space, that etiology of the radiographic findings would not be consistent with the clinical picture as described. Given the fact that the chest radiograph performed yesterday was clear, this represents a sudden process. Therefore, total left lung atelectasis is the diagnosis.

Positioning the patient in the left lateral decubitus position is incorrect, because it would not fix the problem, and may actually worsen the hypoxemia by increasing dependent blood flow to the left lung. Inserting a tube or needle in the pleural space would also not correct the problem.

The patient needs aggressive pulmonary toilet and, if unsuccessful in re-expanding the lung, a therapeutic bronchoscopy will be required. The patient likely has copious secretions that need to be removed in order to improve the aeration of the left lung.

#### **REFERENCES (15)**

Duggan M, Kavanagh BP. Atelectasis in the perioperative patient. Curr Opin Anaesthesiol. 2007; 20:37-42.

Duggan M, Kavanagh BP. Pulmonary atelectasis: A pathogenic perioperative entity. *Anesthesiology*. 2005; 102:838-854.

Hagberg C, Georgl R, Krler C. Complications of managing the airway. Best Pract Res Clin Anaesthesiol. 2005; 19:641-659.

Qureshi NR, Gleeson FV. Imaging of pleural disease. Clin Chest Med. 2006; 27:193-213.

RATIONALE (16)

Answer: B

This patient has an endotracheal tube obstruction. The key to the identification of this problem relates to an understanding of the components of peak inspiratory pressure and inspiratory plateau pressure. Peak inspiratory pressure is created by the pressure required to overcome resistance in the endotracheal tube and airways, as well as the elastance of the lung parenchyma, the pleural space and the chest wall. Inspiratory plateau pressure is pressure generated to overcome the elastance of the lung parenchyma, pleural space and chest wall and is not related to endotracheal tube and airways resistance. In this patient, it is noted that peak inspiratory pressure is steadily rising between day 1 and day 4, whereas inspiratory plateau pressure remains constant. Therefore, this is compatible with increasing endotracheal tube obstruction, in this case due to inspissate mucous.

Flash pulmonary edema and tension pneumothorax would be associated with increases in both peak inspiratory pressure as well as inspiratory plateau pressure. Pulmonary embolus may not produce any changes in inspiratory pressure, but if it produces significant parenchymal lung problems, it would be associated with the same pattern as flash pulmonary edema tension pneumothorax.

#### **REFERENCES (16)**

- Boque MC, Gualis B, Sandiumenge A, Rello J. Endotracheal tube intraluminal diameter narrowing after mechanical ventilation: use of acoustic reflectometry. *Intensive Care Med* 2004;30:2204-2209.
- Dieter RA Jr. Endotracheal tube obstruction, recognition, and management. Mil Med 1990;155:A11.
- Guttmann J, Eberhard L, Haberthur C, et al. Detection of endotracheal tube obstruction by analysis of the expiratory flow signal. *Intensive Care Med* 1998;24:1163-1172.
- Kawati R, Lattuada M, Sjostrand U, et al. Peak airway pressure increase is a late warning sign of partial endotracheal tube obstruction whereas change in expiratory flow is an early warning sign. *Anesth Analog* 2005;100:889-893.
- Sprung J, Bourke DL, Harrison C, Barnas GM. Endotracheal tube and tracheobronchial obstruction as causes of hypoventilation with high inspiratory pressures. *Chest* 1994;105:550-552.
- Stoen R, Smith-Erichesen N. Airway obstruction associated with an endotracheal tube. *Intensive Care Med* 1987;13:295-296.

RATIONALE (17-18)

Answers: 17-A; 18-D

This patient has hypokalemia induced by renal tubular acidosis. The cause of the renal tubular acidosis in this patient was the inhalation of toluene. Renal tubular acidosis is a group of disorders of tubular function that lead to hyperchloremic metabolic acidosis in the presence of well-preserved glomerular function. Toluene produces a classic distal renal tubular acidosis (type 1 renal tubular acidosis) including hypokalemia, hyperchloremic acidosis, low urinary ammonium excretion (positive urine anion gap, low urine ammonia), and inappropriately high urine pH (pH >5.5 in the presence of significant metabolic acidemia).

The potential iatrogenic complication in this patient that is of the most concern is the effect on the potassium of the initial mechanical ventilation settings. This patient has extremely low potassium in the presence of significant acidemia, as well as an elevated PAco<sub>2</sub>. If, after intubation and initiation of mechanical ventilation, the PAco<sub>2</sub> is allowed to be significantly lower than normal (as often happens following initiation of mechanical ventilation), it would produce life-threatening hypokalemia, as the rise in pH would produce a further drop in potassium.

#### **REFERENCES (17-18)**

Bagga A, Bajpai A, Menon S. Approach to renal tubular disorders. *Indian J Pediatr* 2005;72:771-776.
Frassetto LA, Morris RC Jr, Sebastian A. A practical approach to the balance between acid production and renal acid excretion in humans. *J Nephrol* 2006;19(suppl 9):S41-S45.
Laing CM, Unwin RJ. Renal tubular acidosis. *J Nephrol* 2006;19(suppl 9):S46-S52.
Ring T, Frische S, Nielsen S. Clinical review: Renal tubular acidosis—a physicochemical approach. *Crit Care* 2005;9:573-580.

RATIONALE (19)

Answer: A

Heliox is a mixture of helium and oxygen. Helium is less dense in air and will decrease resistance in large airways, improving air-flow limitation by decreasing turbulent flow. Ventilation and CO<sub>2</sub> elimination are an end result. Intrinsic positive end expiratory pressure may also decrease pulseless paradoxus, while peak expiratory flow rate increases. The use of heliox may improve physiology until bronchodilators and corticosteroids can relieve bronchospasm and avoid intubation. However, there have been no large controlled trials that demonstrate any definitive impact on outcome.

Pregnancy is not a contraindication to use of heliox. In general, the risk of inadequate therapy for asthma outweighs the risk of traditional asthma medications. Bronchodilators and corticosteroids should be administered to all pregnant patients with acute asthma exacerbation of this degree. However, epinephrine therapy is not recommended in pregnancy.

Heliox is contraindicated in patients requiring high flows of oxygen. Heliox works best when the admixture is 70:30 (30% oxygen) and may be helpful at 60:40 admixture. Patients requiring more than 30%-40% oxygen to maintain adequate saturation should not receive heliox therapy because of the risk of hypoxemia.

#### **REFERENCES (19)**

- Kim IK, Saville AL, Sikes KL, Corcoran TE. Heliox-driven albuterol nebulization for asthma exacerbations: an overview. *Respir Care* 2006;51:613-618.
- Hess DR, Fink JB, Venkataraman ST, et al. The history and physics of heliox. *Respir Care* 2006;51:608-612.
- Wigmore T, Stachowski E. A review of the use of heliox in the critically ill. *Crit Care Resusc* 2006;8:64-72.
- Jacobs M, Reid C, Butler J. Best evidence topic report. Use of heliox for acute asthma in the emergency department. *Emerg Med J* 2004;21:498-499.
- Reubin AD, Harris AR. Heliox for asthma in the emergency department: a review of the literature. Emerg Med J 2004;21:131-135.

RATIONALE (20)

Answer: B

A patient with abnormalities of the chest wall typically shows a decrease in vital capacity, total lung capacity, and functional residual capacity, with little or no change in residual volume. The diffusion capacity, referenced to alveolar volume, is normal. Reduced vital capacity occurs due to the altered pressure volume characteristics of the chest wall and decreased lung compliance.

The ideal candidate for electrophrenic pacing in diaphragm failure would have normal lungs, normal diaphragms, and viable phrenic nerves; a quadriplegic patient with a high spinal cord lesion fulfills these criteria.

Hypothyroidism respiratory failure is characterized by several pathophysiology processes. These include decreased ventilatory responsiveness to both hypoxemia and hypercapnia, increased mechanical load to the diaphragm due to obesity, and a respiratory myopathy.

Respiratory alternans is a term describing intermittent paradoxic inward diaphragm motion lasting for several breaths. This pattern of muscle use allows intermittent diaphragm rest. Although tidal volumes may be smaller during periods of diaphragm inactivity, the definition of respiratory alternans does not depend on tidal volume differences.

## **REFERENCES (20)**

El Solh AA, Ramadan FH. Overview of respiratory failure in older adults. *J Intensive Care Med* 2006;21:345-351.

Forte P, Mazzone M, Portale G, et al. Approach to respiratory failure in emergency department. Eur Rev Med Pharmacol Sci 2006;10:135-151.

Mehta S. Neuromuscular disease causing acute respiratory failure. Respir Care 2006;51:1054-1061.

RATIONALE (21)

Answer: D

Despite significant improvement in oxygenation and no evidence of toxic side effects, inhaled nitric oxide has not been demonstrated to improve clinical outcome variable such as ICU length of stay, hospital length of stay or mortality.

A recent study by the National Heart, Lung and Blood Institute Acute Respiratory Distress Syndrome Clinical Trials Network compared two fluid management strategies in patients with acute respiratory distress syndrome (ARDS), a conservative strategy group and a liberal strategy group. The conservative strategy group targeted a CVP of <4 or pulmonary artery occlusive pressures of <8, as long as there was hemodynamic stability and no evidence of tissue hypoperfusion. In this group there was improved lung function and a shortening of the duration of mechanical ventilation and intensive care, without increasing nonpulmonary organ failures. However, there was no proven survival benefit.

Despite an initial randomized prospective clinical trial assessing the potential benefit of prone positioning in ARDS that failed to show benefit, a recent study that increased the average hours per day of prone positioning to 17 hours a day (from the previous study's average of 8 hours a day) supports its usage. However, this was achieved only through a multivariate analysis.

The Acute Respiratory Distress Syndrome Clinical Trials Network's first trial comparing 6 mL/kg predicted body weight and 12 mL/kg predicted body weight showed significant improvement in mortality with 6 mL/kg. This protocol also called for continued lowering of the tidal volume in the 6 mL group to obtain an inspiratory plateau pressure <30, as would apply in the case.

#### **REFERENCES (21)**

- Comparison of two fluid-management strategies in acute lung injury. The National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network, N Engl J Med 2006;354:2564-2575.
- Efficacy and Safety of Corticosteroids for persistent acute respiratory distress syndrome. The National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network. N Engl J Med 2006;354:1671-1684.
- Mancebo J, Fernández R, Blanch L, et al. A multicenter trial of prolonged prone ventilation in severe acute respiratory distress syndrome. *Am J Respir Crit Care Med* 2006;173:1233-1239.
- Pulmonary-artery versus central venous catheter to guide treatment of acute lung injury. The National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network, *N Engl J Med* 2006;354:2213-2224.

RATIONALE (22)

Answer: D

Diagnostic accuracy may be improved by gram staining of polymorphonuclear leukocytes and macrophages, and by careful examination of the morphology of any bacteria found to be present in the tracheal aspirates. In a patient without a recent change in antibiotics, a negative tracheal aspirate (absence of bacterial or inflammatory cells) has a strong negative predictive value (94%) for ventilator-associated pneumonia. A reliably performed Gram stain of tracheal aspirates has been shown to result in a low incidence of inappropriate therapy when used to guide initial antibiotic therapy. Tracheal aspirate cultures almost always contain the pathogens found in invasive quantitative cultures of the lower respiratory tract.

The selection of an initial antibiotic therapy is based on risk factors for specific pathogens, modified by knowledge of local patterns of antibiotic resistance in organism prevalence. Therapy is modified on the basis of the clinical response on days 2 and 3; as well as on the findings on semiquantitative cultures of the lower respiratory tract secretions. Use of clinical findings plus semiquantitative cultures of tracheal aspirates is called "clinical strategy."

A bacteriologic strategy uses quantitative cultures of the lower respiratory secretions (endotracheal aspirates and bronchoalveolar lavage or photosynthetic bacteria specimens collected with or without a bronchoscope) to define both the presence of pneumonia and the etiologic pathogen. Growth above a threshold concentration is required to diagnose ventilator-associated or hospital-associated pneumonia and to determine the causative microorganisms. The bacteriologic approach emphasizes preventing excessive use of antibiotics by trying to separate colonizing from infecting pathogens. Use of the bateriologic method has consistently lead to finding fewer microorganism growing above the diagnostic threshold, as compared with the use of nonquanitative cultures of tracheal aspirates.

Quantitative culture data have proven highly effective in diagnosing pneumonia, especially in patients with a lower equivocal clinical suspicion of infection. Bronchoscopic bronchoalveolar lavage studies have typically used a diagnostic threshold of 10<sup>4</sup> or 10<sup>5</sup> colony-forming unit (CFU)/mL, while quantitative culture data of photosynthetic bacteria samples have used a diagnostic threshold of 10<sup>3</sup> CFU/mL. The presence of bacteria on an unspun bronchoalveolar lavage specimen is strongly associated with a lower respiratory tract infection. However, if there is a high pretest probability of pneumonia, or if the patient shows evidence of sepsis, prompt therapy is required whether bacteria are found on microscopic examination of the lower respiratory tract secretions or not.

#### **REFERENCES (22)**

- American Thoracic Society Documents. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. *Am J Respir Crit Care Med* 2005;171:388-416.
- Blot F, Raynard B, Chachaty E, Tancrede C, Antoun S, Nitenberg G. Value of Gram stain examination of lower respiratory tract secretions for early diagnosis of nosocomial pneumonia. Am J Respir Crit Care Med 2000;162:1731-1737.
- The Canadian Critical Care Trials Group. A randomized trial of diagnostic techniques for ventilator-associated pneumonia. N Engl J Med 2006;355:2619-2630.
- Cook D, Mandell L. Endotracheal aspiration in the diagnosis of ventilator-associated pneumonia. *Chest* 2000;117:195S-197S.
- Fagon JY, Chastre J, Wolff M, et al. Invasive and noninvasive strategies for management of suspected ventilator-associated pneumonia: a randomized trial. *Ann Intern Med* 2000;132:621-630.
- Fartoukh M, Maitre B, Honore S, Cerf C, Zahar JR, Brun-Buisson C. Diagnosing pneumonia during mechanical ventilation: the clinical pulmonary infection score revisited. *Am J Respir Crit Care Med* 2003;168:173-179.
- Kirtland SH, Corley DE, Winterbauer RH, et al. The diagnosis of ventilator-associated pneumonia: a comparison of histologic, microbiologic, and clinical criteria. *Chest* 1997;112:445-4457.
- Rumbak MJ, Bass RL. Tracheal aspirate correlates with protected specimen brush in long-term ventilated patients who have clinical pneumonia. *Chest* 1994;106:531-534.
- Torres A, Ewig S. Diagnosing ventilator-associated pneumonia. N Engl J Med 2004;350:433-435.

RATIONALE (23)

Answer: D

In this patient, the pulmonary artery tracing and balloon inflation demonstrate a partial occlusion. Careful examination of the tracing when aligned with the ECG monitor strip would reveal the absence of the characteristic a- and b- waves of a pulmonary artery (PA) occlusion pressure. This is a dampened PA tracing obtained with partial occlusion pressure. However, the partial occlusion does produce a small drop in pressure, and when combined with the dampening, can easily be misinterpreted as a PA occlusion pressure.



This is particularly problematic in patients with pulmonary hypertension, where the partial occlusion will produce a drop in the pressure tracing, as is demonstrated in this case. Partial occlusion should be suspected in a patient with known PA hypertension and with a clinical examination that does not support high pulmonary capillary pressure, as is demonstrated in this case. The degree of pulmonary artery hypertension is underestimated (the gradient between the PA diastolic and the partial occlusion pressure), while the pulmonary capillary pressure and left ventricular end-diastolic pressure is overestimated.

When partial occlusion is suspected, the initial approach is to withdraw the catheter and attempt to obtain the occlusion tracing with a decreased balloon inflation, ie, 1 mL instead of 1.5 mL. In this case, the slight advancement of the fully inflated catheter yielded the "best" occlusion pressure estimate. When the occlusion pressure was obtained in this patient with 1 mL of air, the actual correct pulmonary artery occlusion pressure of 10 mm Hg was obtained.

# **REFERENCES (23)**

Harvey RM, Enson Y. Pulmonary vascular resistance. Adv Intern Med 1969;15:73-93.

Kaltman AJ, Herbery WH, Conroy RK, et al. The gradient in pressure across the pulmonary vascular bed during diastole. *Circulation* 1966;34:377-384.

Leatherman JW, Shapiro RS. Overestimation of pulmonary artery occlusion pressure in pulmonary hypertension due to partial occlusion. *Crit Care Med* 2003;31:93-97.

Morris AH, Chapman RH, Gardner RM. Frequency of wedge pressure errors in the ICU. Crit Care Med 1985;13:705-708.

RATIONALE (24)

Answer: A

This patient has a unilateral acute infiltrative lung disease with severe hypoxemia. The patient remains severely hypoxemic, despite optimization of mechanical ventilator support, including aggressive PEEP therapy. In unilateral infiltrative lung disease with severe hypoxemia, there is no tried and true therapeutic option. Positive end expiratory pressure (PEEP) may be useful in the affected lung, but, at the same time, the application of the PEEP to the uninvolved lung may overinflate and divert blood flow to the involved lung, exacerbating the hypoxemia based on the distribution of the flow. It should, nevertheless, be tried.

The same thought process applies to recruitment maneuvers as it does for PEEP therapy. Prone positioning may be of utility, although there are no controlled studies in this area. However, because prone positioning exerts beneficial effects on oxygenation in bilateral lung disease, it could be potentially useful in unilateral lung disease.

Inhaled nitric oxide offers a potential utility by preferentially vasodilating the uninvolved lung vasculature, diverting blood away from the involved lung. Positioning is most likely to be useful. The involved lung is placed dependently to better match the gravitational flow of blood with the uninvolved lung. In this case, that would be left lung down.

#### **REFERENCES (24)**

Bosma K, Fanelli V, Ranieri VM. Acute respiratory distress syndrome: update on the latest developments in basic and clinical research. *Cur Opin Anaesthsiol* 2005;18:137-145.

Brower RG, Morris A, MacIntyre N, et al. Effects of recruitment maneuvers in patients with acute lung injury and acute respiratory distress syndrome ventilated with high positive end-expiratory pressure. *Crit Care Med* 2003; 31:2592-2597.

Gattiononi L, Tognoni G, Pesenti A, et al. Effect of prone positioning on the survival of patients with acute respiratory failure. *N Engl J Med* 2001;345:568-573.

RATIONALE (25)

Answer: B

A recruitment maneuver, as demonstrated in the ARDSNet ALVEOLI trial, produces a significant improvement in oxygenation. The recruitment maneuver utilized in this trial was 35 cm of continuous positive airway pressure (CPAP) maintained for 30 seconds. Recruitment maneuvers are aborted if BP drops to <90 mm Hg or by >30 mm Hg; the HR increases to  $\ge140$ /min or by >20/min; oxyhemoglobin saturation, as measured by pulse oximetry, decreases to <90% or decreases by  $\ge5\%$ ; or a cardiac dysrhythmia occurrs.

Following the recruitment maneuver, the patient was returned to the previous positive end expiratory pressure (PEEP) setting. Although oxygenation significantly improved, this improvement was short-lived (not >60 min). It should be noted that although sustained inflation maneuvers are utilized to open a previously collapsed lung as part of the recruitment process, PEEP is considered as the mainstay for maintaining the newly recruited lung. Therefore, the use of a recruitment maneuver without increasing the PEEP setting may be counterproductive for the long-term.

In addition, most literature reports have utilized more aggressive inflation maneuvers, typically 40 cm H<sub>2</sub>O PEEP for 40 seconds, or even greater. The ARDSNet low versus conventional tidal volume trial compared 6 mL/kg/IBW versus 12 mL/kg/IBW; 6 mL was demonstrated to improve survival, as compared with 12 mL. Controversy exists as to whether intermediate levels of tidal volume might be as good as or even potentially better than 6 mL. This controversy continues. The ALVEOLI trial compared the use of PEEP settings based on Fio<sub>2</sub> per the first ARDSNet low versus conventional tidal volume trial, with higher PEEP settings at each Fio<sub>2</sub> level. Six mL/kg/IBW tidal volume was used for both groups. There was a significant improvement in Pao<sub>2</sub> with the higher PEEP, with a decrease in Fio<sub>2</sub> requirements. However, there was no difference in outcome. One consistent finding across all ARDSnet trials is a correlation of the inspiratory plateau pressure with outcome.

### **REFERENCES (25)**

Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volume as compared with traditional tidal volume for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 2000;342:1301-1308.

The ARDS Clinical Trials Network; National Heart, Lung, and Blood Institute; National and Institute of Health. Effects of recruitment maneuvers in patients with acute lung injury and acute respiratory distress syndrome ventilated with high positive end-expiratory pressure. Critical Care Medicine 2003;31:2592-2597.

The National Heart, Lung, and Blood Institute ARDS Clinical Trials Network. Higher versus lower positive end-expiratory pressures in patients with acute respiratory distress syndrome. N Engl J Med 2004;351:327-336.

RATIONALE (26)

Answer: B

Intubation and reintubation should be avoided, if possible, as it increases the risk of ventilator-acquired pneumonia (VAP). Noninvasive ventilation should be used whenever possible as it decreases the incidence of hospital-acquired pneumonia when compared to endotracheal intubation. Continuous aspiration of subglottic secretions has been demonstrated to reduce risk of early onset VAP. An endotracheal tube cuff pressure of slightly above 20 cm H<sub>2</sub>O prevents leakage of bacterial pathogens around the cuff. Patients who are kept in the semirecumbent position (30-45°), rather than supine positive, have a decreased incidence of VAP.

#### **REFERENCES (26)**

- American Thoracic Society; Infectious Disease Society of America: Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. *Am J Respir Crit Care Med* 2005;171:388-416.
- Brochard L, Mancebo J, Wysocki M, et al. Non invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. *N Engl J Med* 1995;333:817-822.
- Rello J, Sonora R, Jubert P, et al: Pneumonia in intubated patients; role of respiratory airway care. Am J Respir Crit Care Med 1996; 154:111-115.
- Valles J, Artigas A, Rello J, et al: Continuous aspiration of subglottic secretions in preventing ventilator-associated pneumonia. *Ann Intern Med* 1995;122:179-186.

# SECTION 13: Renal/Fluids/Electrolytes

# SECTION 13: RENAL/FLUIDS/ELECTROLYTES

Instructions: For each question, select the most correct answer.

1. You are called to the oncology ward to evaluate a 60-year-old female with a known history of lung cancer who has developed a new-onset seizure. The patient was given IV lorazepam, and her seizures resolved. On examination, vital signs are as follows: BP 120/70 mm Hg, HR 95/min, temperature 36.5°C (97.7°F), and RR 16/min. Physical examination is significant for pupils that are equal and reactive bilaterally; patient is lethargic, not responding to verbal stimuli but will respond to pain moving all extremities. Lung examination reveals decreased breath sounds on the left with clear auscultation on the right side. A stat noncontrast CT scan of the head shows no abnormalities. Laboratory data are as follows: sodium 104 mEq/L, potassium 4.8 mEq/L, chloride 85 mEq/L, bicarbonate 20 mEq/L, blood urea nitrogen 10 mg/dL, creatinine 0.5 mg/dL, and serum glucose is 100 mg/dL. Urinalysis reveals a pH of 5.0, with a specific gravity of 1.000.

Which of the following is the most appropriate treatment for this patient?

- A. Demeclocycline
- B. Free-water fluid restriction
- C. Dexamethasone
- D. Phenytoin
- E. 3% saline solution

2. A 25-year-old male with insulin-dependent diabetes mellitus is admitted to the ICU with uncontrolled diabetes. He has a 1-day history of nausea, vomiting, and diarrhea, and he withheld insulin because of poor oral intake. His BP is 100/60 mm Hg, HR is 104/min, RR is 28/min, and temperature is 37.22°C (99°F). Physical examination reveals dry mucous membranes and mild diffuse abdominal tenderness. Laboratory examinations reveal sodium 135 mmol/L, potassium 3.5 mmol/L, chloride 110 mmol/L, CO<sub>2</sub> 8 mmol/L, BUN 22 mg/dL, creatinine 1.2 mg/dL, albumin 4.1 g/dL, and glucose 350 mg/dL. Arterial blood gas measurements are (Fio<sub>2</sub> 0.21) pH 7.25, Paco<sub>2</sub> 20 mm Hg, and Pao<sub>2</sub> 95 mm Hg; serum ketones are present at a 1:8 dilution.

Which one of the following is the best explanation for the acid-base abnormality in this patient?

- A. Diabetic ketoacidosis
- B. Diabetic ketoacidosis with hyperventilation
- C. Diabetic ketoacidosis and diarrhea
- D. Diabetic ketoacidosis and nausea/vomiting
- E. Laboratory error
- 3. A 45-year-old male was admitted to the ICU with uncontrolled hypertension. He was first diagnosed with hypertension 5 days earlier and started on enalapril 10 mg daily. He was taking no other medications. On the day of admission, he developed an intractable headache along with nausea and 2 episodes of vomiting. His BP is 260/120 mm Hg, without orthostatic changes. Laboratory studies revealed sodium 130 mmol/L, potassium 3.0 mmol/L, chloride 90 mmol/L, HCO<sub>3</sub> 32 mmol/L, blood urea nitrogen 14 mg/dL, and creatinine 0.8 mg/dL. Arterial blood gas measurements while receiving room air are pH 7.47, Paco<sub>2</sub> 45 mm Hg, Pco<sub>2</sub> 74 mm Hg, and a random urine chloride is 30 mmol/L.

Which one of the following best explains the acid base abnormality in this patient?

- A. Primary hypoventilation due to hypertensive encephalopathy
- B. Vomiting
- C. Primary hypoventilation and vomiting
- D. Mineralocorticoid excess
- E. Laboratory error

4. A 45-year-old alcoholic man (70 kg) is brought to the hospital with altered mental status. He had no focal neurologic deficits, and a CT scan of the head was negative for bleeding or masses. He was admitted to the ICU because of a sodium level of 115 mmol/L. Vital signs are BP 110/74 mm Hg, HR 96/min, RR 14/min, and temperature 37.22°C (99°F). No orthostasis is noted. He was lethargic but followed simple commands. Other laboratories included potassium 3.1 mmol/L, chloride 86 mmol/L, HCO<sub>3</sub> 22 mmol/L, blood urea nitrogen 8 mg/dL, creatinine 0.5 mg/dL, glucose 106 mg/dL, and ethanol 180 mg/dL. Urinalysis showed a specific gravity 1.010, pH 6, and negative blood and glucose results. Urine osmolarity measurement has been requested and is pending.

Which one of the following therapies is most appropriate pending further laboratory results?

- A. Free water restriction
- B. 1 L normal saline solution over 1 hour
- C. 1 L normal saline solution over 1 hour + 40 mg furosemide IV
- D. 100 mL 3% saline solution over 1 hour
- 5. You are consulted to evaluate a 65-year-old male with worsening mental status. The patient was admitted to the hospital 2 days ago for an elective transurethral resection of the prostate. He was otherwise in good health and taking no medications. On admission, laboratory data, including a complete blood count, electrolytes, blood urea nitrogen, and creatinine, were within normal ranges. His pre-admission medications include metoprolol, an angiotension-converting enzyme (ACE) inhibitor, and a statin. On the first postoperative day, the patient complained of headache and nausea. Symptoms progressed. The patient became lethargic and was transferred to the ICU.

On examination, the patient was afebrile, with a HR of 90/min, BP 150/90 mm Hg, and RR 15/min. He was stuporous, unable to follow commands, and withdrew all 4 extremities to pain. Subsequently, the patient developed a generalized tonic-clonic seizure.

Laboratory examination at this point revealed a normal complete blood count, sodium 114 mEq/L, potassium 3.8 mEq/L, chloride 78 mEq/L, HCO<sub>3</sub> 20 mEq/L, blood urea nitrogen 18 mg/dL, and creatinine 1.2 mg/dL.

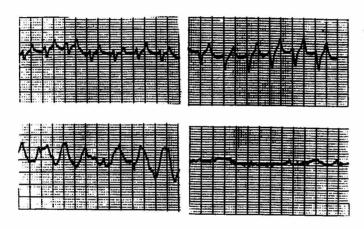
The most likely cause of this patient's hyponatremia is which of the following?

- A. Metoprolol
- B. Isotonic saline solution
- C. Hypotonic irrigating solution
- D. Furosemide

- 6. Which one of the following is not a cause of chloride-responsive metabolic alkalosis?
  - A. Excess use of loop diuretics
  - B. Pyloric stenosis due to duodenal ulcer
  - C. Villous adenoma diarrhea
  - D. Posthypercapnic metabolic alkalosis
  - E. Primary aldosteronism
  - 7. Which one of the following is the most likely cause of the following progression of rhythm strips?

**First** 

Second



Third

Fourth

- A. Hypocalcemia
- B. Hyperkalemia
- C. Hypomagnesemia
- D. Hypercalcemia

- 8. A 28-year old female with insulin-dependent diabetes mellitus is admitted to the ICU with diabetic ketoacidosis. Electrolytes are Na 135 mEq/L, Cl 100 mEq/L, bicarbonate 8 mEq/L, and glucose 500 mg/dL; arterial blood gas reveals pH 7.22, Paco<sub>2</sub> 20 mm Hg. Serum ketones are positive at 1:8. Which one of the following is most correct concerning this patient's metabolic status?
  - A. A primary respiratory alkalosis is present
  - B. There is no clinically significant free water deficit
  - C. A nonanion gap acidosis is present
  - D. The serum ketone level supports a cause of anion gap in addition to ketoacidosis
- 9. For each of the following hypotensive patients, aggressive crystalloid therapy has failed to normalize blood pressure (BP).

The administration of hydroxyelthyl starch (HES) should be avoided in which one of the following patients?

- A. A 24-year-old male with chronic renal failure on hemodialysis who is admitted to the intensive care unit with septic shock
- B. A 36-year-old female with a platelet count of 20,000
- C. A 68-year-old female admitted to the intensive care unit with anaphylactic shock
- D. A 29-year-old male with Hodgkin's lymphoma admitted for consolidation chemotherapy
- E. A 47-year-old male with acute respiratory distress syndrome (ARDS)
- 10. Which one of the following is most likely to cause hemodialysis-induced hypotension?
  - A. High Na+dialysate
  - B. Bicarbonate-buffered dialysate
  - C. 35°C (95°F) dialysate
  - D. Low calcium dialysate
- 11. Which one of the following electrocardiographic changes is least likely to occur with hypokalemia?
  - A. ST-T segment depression
  - B. T wave inversion
  - C. AV blocks (2<sup>nd</sup> or 3<sup>rd</sup> degree)
  - D. Premature ventricular beats
  - E. Appearance of U waves

- 12. Which one of the following findings is most likely to be present in a patient with severe magnesium deficiency?
  - A. Respiratory depression
  - B. Bradycardia
  - C. Tetany
  - D. Hypotension
  - E. Loss of patellar reflex
- 13. A 45-year-old male presents with increased lethargy and severe headache. A CT scan of the brain with no contrast reveals a subarachnoid bleed. Patient undergoes cerebral angiogram with coiling of an aneurysm in the left middle cerebral artery. Five days postprocedure the patient's sodium drops to 122 mEq/L.

Which one of the following statements is most correct regarding the differentiation of the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and cerebral salt wasting (CSW) in this case?

- A. Distinction between SIADH and CSW is not clinically relevant, since the treatment is the same
- B. Measurement of urine sodium concentration will best differentiate SIADH and CSW
- C. Assessment of extracellular fluid (ECF) volume will best differentiate SIADH and CSW
- D. Measurement of urine concentration and osmolality will best differentiate SIADH and CSW
- E. Distinction between SIADH and CSW cannot be done because they share the same underlying pathophysiology
- 14. Which one of the following is least compatible with oliguric acute tubular necrosis?
  - A. Urine osmolality of 280 mOsm
  - B. Urine sodium concentration of 15 mg/L
  - C. Fractional excretion of sodium of 2.5%
  - D. Urine/plasma urea ratio of 2
  - E. Urine/plasma creatine ratio of 18

# SECTION 13: RENAL/FLUIDS/ELECTROLYTES

#### **ANSWERS:**

1-E; 2-C; 3-D; 4-A; 5-C; 6-E; 7-B; 8-B; 9-B; 10-D; 11-C; 12-C; 13-C; 14-B

RATIONALE (1)

Answer: E

The differential diagnosis of new onset seizures with known lung cancer includes brain metastasis and severe electrolyte abnormalities and most importantly hyponatremia and hypercalcemia. This patient presentation is most consistent with seizures secondary to severe hyponatremia. This patient has symptomatic hyponatremia secondary to the syndrome of inadequate antidiuretic hormone secretion (SIADH). SIADH can be associated with various clinical disorders including lung cancer. Aggressive treatment is indicated for severe complications from hyponatremia, such as seizures. This patient should be started on 3% saline solution and might require furosemide diuresis. Less aggressive management, such as free-water restriction, should be reserved for less severe cases of hyponatremia, where only mild neurologic symptoms are present. Demeclocycline blocks peripheral actions of antidieretic hormone and can also be utilized for milder cases of SIADH. Dexamethasone is usually recommended for cerebral edema secondary to central nervous system metastasis, which, in this patient, is not evident. Phenytoin can be used for seizures secondary to metastasis. However, in this patient, correction of the sodium will resolve the seizures.

## **REFERENCES (1)**

Berl T, Taylor J. Disorders of water balance. In: Fink MP, Abraham E, Vinvent JL, and Kochanek PM, ed. Textbook of Critical Care. 5th ed. Philadelphia, PA: Elisevier; 2005.
Palevsky PM, Bhagrath R, Greenberg A. Hypernatremia in hospitalized patients. Ann Int Med 1996; 124:197-203.

RATIONALE (2)

Answer: C

The laboratory measurements reveal acidemia with decreased bicarbonate concentration, indicative of a metabolic acidosis. The degree of respiratory compensation is adequate (expected Pco, in a simple metabolic acidosis =  $[1.5 \text{ x HCO}_3] + 8 \pm 2$ ), thereby excluding primary hyperventilation related to anxiety. The anion gap is increased, and in view of the hyperglycemia and ketonemia, the patient has diabetic ketoacidosis. However, the anion gap is 17 mmol/L, and this is 5 mmol/L higher than the upper limit of normal of 12 mmol/L. In a "pure" anion gap metabolic acidosis, the decrease in serum bicarbonate should approximate the increase in anion gap. Thus, the expected bicarbonate in this patient is 19 mmol/L (normal bicarbonate of 24 mmol/L, minus the change in anion gap of 5 mmol/L). However, the actual bicarbonate of 8 mmol/L is considerably lower than anticipated, reflective of a change in bicarbonate of 16 mmol/L. The difference between the change in bicarbonate and the change in anion gap is known as the "delta gap" and normal values are  $0 \pm 6$ . Because the serum bicarbonate is much lower than expected for a simple anion gap metabolic acidosis, the acid-base status of this patient is best described as a mixed anion gap metabolic acidosis (diabetic ketoacidosis) with a superimposed nonanion gap (hyperchloremic) metabolic acidosis. From the history, diarrhea is the most likely explanation for the hyperchloremic metabolic acidosis. The serum bicarbonate is consistent with the pH and Pco, (based on the Henderson-Hasselbach equation); therefore, a laboratory error does not account for the acid-base abnormalities.

#### **REFERENCES (2)**

- Kellum JA. Diagnosis and treatment of acid-base disorders. In: Grenvik AG, Ayres SM, Holbrook PR, Shoemaker WC, eds. Critical Care. 4th ed. Philadelphia, PA: WB Saunders; 2000:834-853.
  Paulsen WD, Gadallah MF. Diagnosis of mixed acid-base disorders in diabetic ketoacidosis. Am J Med Sci 1993; 305:295-300.
- Ring T, Frische S, Nielsen S: Clinical review: Renal tubular acidosis a physicochemical approach. *Crit Care Med* 2005;9:573-580.
- Wrenn K. The delta (D) gap: an approach to mixed acid-base disorders. Ann Emerg Med 1990; 19:1310.

RATIONALE (3) Answer: D

The presence of alkalemia with increased serum bicarbonate indicates a primary metabolic alkalosis. The alkalemia leads to direct suppression of ventilation, and the Paco<sub>2</sub> rises 0.6 to 0.7 mm Hg for each 1-mmol/L increase in serum bicarbonate. Therefore, in this patient, respiratory compensation is appropriate for the metabolic alkalosis. The pH, bicarbonate, and Paco<sub>2</sub> are internally consistent, and therefore, laboratory error does not explain the abnormalities.

The measurement of urine chloride concentration is useful to determine the etiology of metabolic alkalosis. Although this patient has a history of vomiting, the presence of hypertension, hypokalemia, normal blood urea nitrogen, and a urine chloride concentration >20 mmol/L suggests mineralocorticoid excess as the etiology. The use of enalapril will tend to increase serum potassium, making the hypokalemia in this patient even more significant. Vomiting, nasogastric suction, postdiuretic use, and posthypercapnic state are associated with chloride responsive metabolic alkalosis with urine chloride concentration <15 mmol/L. In contrast, mineralocorticoid excess, current diuretic administration, milk-alkali syndrome, Bartter syndrome, and severe hypokalemia are associated with urine chloride >20 mmol/L. In this patient, the presence of hypertension and hypokalemia suggest mineralocorticoid excess, as seen in primary hyperaldosteronism or hypercortisolism, exogenous corticosteroid use, or licorice abuse.

#### **REFERENCES (3)**

- Adrogué HJ, Madias NE. Management of life-threatening acid-base disorders. N Engl J Med 1998; 338:107-111.
- Dubose TD: Acid-base disorders. In: Brenner BM, ed. Brenner and Rector's The Kidney. 6th ed. Philadelphia, PA: WB Saunders; 2000:925-997.
- Kamel KS, Ethier JH, Richardson RM, et al. Urine electrolytes and osmolality: when and how to use them. Am J Nephrol 1990; 10:89-102.

RATIONALE (4)

Answer: A

This patient appears to have euvolemic hyponatremia based on vital signs, lack of orthostasis, and laboratory values. The differential includes syndrome of inappropriate secretion of antidiuretic hormone (SIADH), primary polydipsia, and hypothyroidism. Although it is helpful to compare the urine osmolarity with serum osmolarity for the diagnosis of SIADH, the urine specific gravity in this patient suggests the presence of primary polydipsia. The urine specific gravity indicates a near maximally dilute urine. Although the patient has mild alteration of mental status, this may be due to alcohol intoxication alone. Observation and water restriction in this patient, with less serious symptoms, will quickly confirm the diagnosis. Once removed from the source of water intake, urine output increases to excrete free water. These patients usually correct the serum sodium to baseline very rapidly without further intervention. Normal saline solution or hypertonic saline solution may be indicated in a patient with SIADH and significant symptoms. An asymptomatic patient requires primarily fluid restriction to slowly increase the serum sodium. Although the rate of correction of hyponatremia is controversial in patients with serious symptoms, the increase should be limited to approximately 8 to 12 mmol/L in the first 24 hours. One option is to accelerate the rate of serum sodium elevation early in the treatment course in the presence of life-threatening symptoms, such as seizures. The change in serum sodium from fluid administration can be estimated from the formula:

#### <u>Infusate Na – Serum Na</u> Total body water + 1

Total body water is estimated as 0.6 for men or 0.5 for women times the body weight in kilograms. Too rapid a correction of serum sodium may result in central nervous system injury (ie, osmotic demyelinating syndrome). None of the fluid options are indicated if the etiology of the hyponatremia is correctly assessed.

# **REFERENCES (4)**

Adrogué HJ, Madias NE. Hyponatremia. N Engl J Med 2000; 342:1581.

Arieff AI. Acid-base, electrolyte, and metabolic abnormalities. In: Parrillo JE, Dellinger RP, eds. Critical Care Medicine. 2nd ed. St. Louis, MO: Mosby, Inc.; 2001:1169-1203.

Fraser CL, Arieff AI. Epidemiology, pathophysiology, and management of hyponatremic encephalopathy. Am J Med 1997; 102:67-77.

Tanneau RS, Henry A, Rouhart F, et al. High incidence of neurologic complications following rapid correction of severe hyponatremia in polydipsic patients. *J Clin Psychiatry* 1994; 55:349-354.

RATIONALE (5)

Answer: C

This patient has developed acute (<48-72 hours) and severe (≤115 mEq/L) hyponatremia with encephalopathy postoperatively. In this scenario, the cause is most likely related to the use of hypotonic irrigating fluids during and after the transurethral resection of the prostate with significant absorption of free water. Neither metoprolol nor the ACE inhibition produce hyponatremia. Furosemide is more likely to produce hyponatremia. Symptoms of hyponatremic encephalopathy are related to the development of brain tissue edema and range from headache, nausea, and lethargy, to seizures, coma, respiratory arrest, and brain herniation. Development of symptoms in hyponatremia is more common with acute changes in serum sodium concentrations. Morbidity and mortality is very high in patients with hyponatremic encephalopathy, and treatment should be instituted emergently. The treatment of choice in this case is hypertonic saline solution to bring the sodium quickly up to a safe level (125 to 130 mEq/L). The rate of correction has been subject of controversy, because rapid corrections of hyponatremia are associated with cerebral myelinolysis and poor outcomes. Many experts recommend avoiding increases >8-10 mEq in a 24hour period. In symptomatic patients, the rate of correction can be 1-2 mEq/L/h for the initial hours of treatment. Furosemide is commonly used in conjunction with hypertonic saline solution in order to prevent pulmonary edema. However, used alone would worsen hyponatremia. Once neurologic symptoms are resolved, isotonic fluids and water restriction can be used to slowly correct sodium levels back to normal. Water restriction alone is indicated for chronic asymptomatic hyponatremia. The recent development of V2-vasopressin receptor antagonists, as therapeutic agents for hyponatremia is promising. These agents are currently undergoing clinical trials and are not available for clinical use.

#### **REFERENCES (5)**

Androgue HJ, Madias NE. Hyponatremia. N Engl J Med 2000; 342:1581-1589.
Gowrishankar M, Lin SH, Mallie JP, et al. Acute hyponatremia in the perioperative period: insights into its pathophysiology and recommendations for management. Clin Nephrol 1998; 50:352-360.
Gross P, Reimann D, Henschkowski J, et al. Treatment of severe hyponatremia: conventional and novel aspects. J Am S Nephrol 2001; 12(suppl 17):S10-S14.

RATIONALE (6)

Answer: E

Urine chloride levels in primary aldosteronism will usually be >20 mEq/L, unless there has been significant chloride restriction. Potassium excretion will tend to be increased, despite low plasma potassium levels. Volume replacement plus chloride will usually not be adequate to correct the problem. All of the other problems listed as choices usually have a urine chloride level <10 mEq/L and will usually respond adequately to correction of the chloride and extracellular fluid volume deficits.

#### **REFERENCES (6)**

Kode SM, Taylor RW. Chloride ion in intensive care medicine. Crit Care Med 1992; 20:227.

Narins RG, Jones ER, Stom MC, et al. Diagnostic strategies in disorders of fluid, electrolyte, and acid-base homeostasis. *Am J Med* 1982; 72:496-520.

Rimmer JM, Gennari FJ. Metabolic alkalosis. J Intensive Care Med 1987; 2:137.

Toto RD. Metabolic acid-base disorders. In: Kokko JP, Tannen RL, ed. *Fluid and Electrolytes*. Philadelphia, PA: WB Saunders; 1986:275-292.

RATIONALE (7)

Answer: B

These rhythm strips show a classic progression of severe life-threatening hyperkalemia. The first sign of increased serum potassium on the ECG is a tenting of the T-wave. Changes associated with further increases in serum potassium include the following:

- A widening of the QRS complex, progressive atrial ventricular conduction blocks;
- A slow idioventricular rhythm;
- An ECG tracing resembling a sine wave
- Either ventricular fibrillation, or as in this case, asystole.

Treatment of severe hyperkalemia includes calcium gluconate to antagonize the effects of the hyperkalemia on the heart, administration of glucose and insulin to push potassium intracellularly, and removal of potassium from the body (Kayexalate and dialysis). Use of sodium bicarbonate is less effective and should be limited to patients that have concurrent significant acidemia. Albuterol 10–20 mg in 4 mL saline by nasal inhalation over 10 minutes can lower the serum potassium significantly.

# **REFERENCES (7)**

Alfonzo AV, Isles C, Geddes C, Deighan C. Potassium disorders: clinical spectrum and emergency management. *Resuscitation* 2006;70:10-25.

Evans KJ, Greenberg A. Hyperkalemia: a review. J Intensive Care Med 2005;20:272-290.

Kraft MD, Btaiche IF, Sacks GS, Kudsk KA. Treatment of electrolyte disorders in adult patients in the intensive care unit. Am J Health Syst Pharm 2005;62:1663-1682.

RATIONALE (8)

Answer: B

There is no free-water deficit present in this patient. An adjustment for the effect of hyperglycemia on the free-water shift from the extravascular space to the intravascular space (and therefore the decrease in Na) can be made by assuming that the serum sodium decreases by 1.6 mg/dL for each 100 mg/dL increase in glucose. Therefore, in this patient, the return of the glucose levels to normal (100 mg/dL), would cause a rise in sodium by 6.4 mEq/L (4 x 1.6). The sodium would be anticipated to be 141 mEq/L after the correction of the elevated glucose period. The sodium is not elevated, and therefore, is not compatible with free-water deficit.

There are two formulas to judge the appropriateness of a hyperventilation response to metabolic acidosis. One says that the expected  $Paco_2$  is equal to the serum bicarbonate x 1.5 + 8+/-2. A more easily remembered calculation is that the hyperventilation response should lower the  $Paco_2$  to approximate the last two digits of the pH, down to a maximum response of 15 mm Hg. In this case, the  $Paco_2$  of 20 mm Hg approximates the last two digits of the pH of 7.22. It appears to be an appropriate response.

Calculation of the anion gap (Na - Cl + Bicarb), reveals the anion gap to be 27 mEq/L. Assuming a normal anion gap of 10 mEq/L, the delta gap is 17. The delta gap identifies the decrease in bicarbonate that would be anticipated to the anion gap acidosis. In this case, the bicarbonate would be expected to be 7 mEq/L (normal bicarbonate of 24 - delta gap of 17). Therefore, nonanion gap acidosis is not present, because the actual serum bicarbonate is 8 mEq/L.

Finally, the serum ketone level measures acetoacetate and acetone and does not measure  $\beta$ -hydroxybutyrate.  $\beta$ -hydroxybutyrate is metabolized first to acetoacetate; acetoacetate is then metabolized to acetone. In the early treatment of ketoacidosis, the serum ketone level does not reflect the magnitude of the anion gap acidosis, which is caused by the sum of  $\beta$ -hydroxybutyrate and acetoacetate.

## **REFERENCES (8)**

Adler SM, Verbalis JG. Disorders of body water homeostasis in critical illness. *Endocrinol Metab Clin North Am* 2006;35:873-894.

Adrogue HJ. Mixed acid-base distrubances. J Nephrol 2006;19(suppl 9):S97-S103.

Barthwal MS. Analysis of arterial blood gases: a comprehensive approach. J Assoc Physicians India 2004;51:573-577.

Gunnerson KJ. Clinical review: the meaning of acid-base abnormalities in the intensive care unit part I: epidemiology. *Crit Care* 2005;9:508-513.

Kaplan LJ, Frangos S. Clinical review: acid-base abnormalities in the intensive care unit: part II. *Crit Care* 2005;9:198-203.

Kellum JA. Clinical review: reunification of acid-base physiology. Crit Care 2005;9:500-507.

Offenstadt G, Das V. Hyponatremia, hypernatremia: a physiological approach. *Minerva Anestesiol* 2006;72:353-356.

Ruth JL, Wassner SJ. Body composition: salt and water. Pediatr Rev 2006;27:181-187.

RATIONALE (9)

Answer: B

Hydroxyethyl starch (HES) is a natural starch of highly branched glucose polymers derived from amlyopectin. Its half-life is approximately 17 days, and following infusion, the circulatory volume expands to a volume slightly greater than the administered volume. Maximum expansion may persist for 24 hours or longer. It is generally commercially available as a 6% solution in 0.9% normal saline. In terms of restoring intravascular volume, its properties are very similar to that of 5% albumin solution.

Although most authors recommend crystalloids such as normal saline as first line therapy for volume resuscitation in shock, HES may be used successfully in patients with hypovolemic/hemorrhagic shock, especially when blood products are not yet available. HES can be considered in septic shock when at least 2L of crystalloid produces no significant improvement in hemodynamic status or when massive amounts of crystalloid are required to maintain intravascular volume. HES has also been used successfully in patients with thermal or anaphylactic shock as well.

Adverse effects of HES include anaphylactoid reactions, precipitation of congestive heart failure or renal failure, hyperamylasemia, and coagulation abnormalities. Although not absolutely contraindicated in patients with heart failure or renal failure, HES must be used with extreme caution in these patients. Use of HES is contraindicated in patients with pre-existing coagulopathy or profound thrombocytopenia.

#### **REFERENCES (9)**

- Vermeulen LC, Ratko TA, Erstad BL, et al. A paradigm for consensus: The University Hospital Consortium Guidelines for the use of albumin, nonprotein colloid, and crystalloid solutions. *Arch Intern Med* 1995;155:373-379.
- de Jonge E, Levi M. Effects of different plasma substitutes on blood coagulation: A comparative review. Crit Care Med 2001;29(6):1261-1267.

RATIONALE (10)

Answer: D

Hypotension occurs in approximately 25% of patients undergoing dialysis. Many cases of dialysis-induced hypotension have been attributed to excessive speed of fluid removal or to the total volume removed during ultrafiltration. The rapid removal of urea is also responsible for many instances of intradialytic hypotension. Urea is in equilibrium across cell membranes. When urea is rapidly removed with dialysis, the intracellular osmotic gradient remains high compared to that of the vascular compartment. This enhances an intracellular shift of fluid to equilibrate the osmotic gradient and hence decreases intravascular volume.

The incidence of intradialytic hypotension due to the choice of dialysate (buffer, Na<sup>+</sup>, and Ca<sup>2+</sup> concentrations and dialysate temperature) and the dialyzer membrane's composition and porosity is less common. Impairment of vasoconstriction, exacerbation of autonomic dysfunction, and declining serum osmolality are thought to be the cause of hypotension when using a hyponatric dialysate. A lowered dialysate Ca<sup>2+</sup> concentration and dialysate temperature of 98.6° F have vasodilatory and cardiodepressant effects. The dialyzer membrane may cause hypotension by complement activation, complement or pyrogen-induced cytokine production, and by kallikrein-bradykinin activation (seen in individuals on angiotensin-converting enzyme inhibitors dialyzed with polyacrylonitrile hemodialyzers). Acetate buffer, previously frequently used for dialysis, is a well know cause of hypotension by its myocardial depressant and vasodilating effects. Dialysate buffer is now rarely a concern because bicarbonate is used instead of acetate.

The ability to respond to dialytic induced hypotension is often compromised by the concomitant medical problems seen in many patients with chronic renal failure. These include left ventricular dysfunction (systolic or diastolic), autonomic dysfunction (due to renal or comorbid disease processes or medications), inappropriate vasodilation (due to eating, sepsis, or medications), and pericardial effusion.

The following are common strategies used for patients at increased risk of intradialytic hypotension:

- Hold antihypertensive medications
- Increase dialysis time and/or frequency of dialysis
- · Reduce blood flow and/or dialyzer surface area
- Increase dialysate Na+ concentration
- Increase dialysate Ca<sup>2+</sup> concentration
- Cool dialysate to 35°C (95°F)
- · Avoid food intake

#### **REFERENCES (10)**

Hoeben H, Abu-Alfa AK, Mahnensmith R, Perazella MA. Hemodynamics in patients with intradialytic hypotension treated with cool dialysate or midodrine. *Am J Kidney Dis* 2002;39:102-7.

Sherman RA. Modifying the dialysis prescription to reduce intradialytic hypotension. Am J Kidney Dis 2001;38(4 Suppl 4):S18-25.

Schreiber MJ Jr. Clinical case-based approach to understanding intradialytic hypotension. Am J Kidney Dis 2001;38(4 Suppl 4):S37-47.

Daugirdas JT. Preventing and managing hypotension. Semin Dial 1994;7:276-283 Meyer MM. Renal replacement therapies. Crit Care Clin 2000;16:29-58.

RATIONALE (11)

Answer: C

Abnormalities in potassium serum concentrations are commonly seen in ICU patients and can lead to serious cardiovascular complications. Hypokalemia is commonly associated with ST-T segment flattening and depression, T wave flattening and inversion, U waves, premature ventricular beats and ventricular tachycardia. High degree AV blocks are seen with severe hyperkalemia. Hyperkalemia can also present with peaked T waves, widening QRS segment, reduced R wave amplitude, disappearance of the P wave and ultimately heart block or development of sine wave. Cardiac arrest is more common with hyperkalemia than hypokalemia, but both abnormalities when present require prompt correction.

# **REFERENCES (11)**

Francis GS. Cardiac complications in the intensive are unit. Clin Chest Med 1999;20:269-85. Arieff AI. Acid-base, Electrolyte, and Metabolic Abnormalities. In: Parrillo JE, Dellinger RP (Eds). Crit Care Med St. Louis, Mosby, Inc., 2001;1169-1203.

RATIONALE (12)

Answer: C

Magnesium plays an important role in membrane stabilization, nerve conduction, ion transport, and calcium channel activity. Alterations in magnesium levels will present with clinical findings reflecting changes in these mechanisms. Severe hypomagnesaemia typically presents with associated hypocalcaemia. The degree of hypocalcaemia is directly correlated to the severity of hypomagnesaemia. Patients will present with neuromuscular hyperexitability and tetany (positive Chvostek and Trousseau signs or spontaneous carpopedal spasm). Other common manifestations of hypomagnesaemia include: depression, psychosis, seizures, vertigo, ataxia, and ventricular arrhythmias. With hypermagnesaemia neuromuscular excitability is depressed. High levels of magnesium are associated with bradycardia, hypotension, respiratory depression, and loss of deep tendon reflexes.

#### **REFERENCES (12)**

Agus MS, Agus ZS. Cardiovascular actions of magnesium. *Crit Care Clin* 2001;17:175-86. Dacey MJ. Hypomagnesemic disorders. *Crit Care Clin* 2001;17:155-73. Weisinger JR, Bellorin-Fort E. Magnesium and phosphorus. *Lancet* 1998;352:391-96.

RATIONALE (13)

Answer: C

Development of hyponatremia is commonly seen in patients with neurosurgical disease. Among the most common causes cited by the literature are cerebral salt wasting (CSW) and the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). Making the distinction between CSW and SIADH has significant therapeutic implications. Treatment in SIADH involves mostly fluid restriction where as in CSW fluid and NaCl replacement are needed. Determination of extracellular fluide (ECF) volume is the primary way to differentiate between these conditions. In SIADH there is an inappropriate secretion of antidiuretic hormone resulting in increased ECF volume and hyponatremia. In CSW impaired handling of sodium by the kidneys, thought to be central in origin, will result in salt wasting and a decrease in ECF volume. Clinical signs of ECF volume depletion include orthostatic hypotension or tachycardia, dry mucous membranes, flat neck veins and weight loss with negative salt/fluid balance. Laboratory manifestations seen with low ECF volume reflect effects of hemoconcentration and include increased BUN, albumin, and hematocrit. Both entities will have increased urine osmolality and concentration. In SIADH this constitutes an inappropriate response and in CSW it is appropriate considering the low ECF volume. Increased urinary sodium and decreased serum uric acid are seen with SIADH and CSW, therefore they are not useful in making a distinction between these 2 entities.

#### **REFERENCES (13)**

Palmer BF. Hyponatremia in a neurosurgical patient: Syndrome of inappropriate antidiuretic hormone secretion versus cerebral salt wasting. Nephrol Dial Transplant 2000;15:262-268.
Harrigan MR. Cerebral salt wasting syndrome: A review. Neurosurgery 1996;38:152-160.
Sivakamur V, Rajshekhar V, Chandy MJ. Management of neurosurgical patients with hyponatremia and natriuresis. Neurosurgery 1994;34:269-274.

RATIONALE (14)

Answer: B

Much information is available to concerning the use of urine osmolality (Uosm), urine to plasma urea ratio (U/PUr), urine to plasma creatine ratio (U/PCr), urinary sodium concentration (UNa) and fractional sodium excretion (FENa) as tests to differentiate acute tubular necrosis (ATN) from prerenal azotemia. Although none of these tests can invariably differentiate between these 2 entities, they do offer useful guidelines in the oliguric patient. Oliguric patients with a urine osmolality less than or equal to 350 mOsm/kg are likely to have ATN, whereas those with an osmolality >500 mOsm/kg are more likely to have prerenal azotemia. In the range of 350 to 500 mOsm/kg, there is less support for a discrimination capability. In prerenal azotemia, urinary sodium is usually < 20 mEq/L. Patients with ATN, chronic renal failure, and chronic hydronephrosis characteristically have a much UNa. There is considerable overlap in the range between 20 and 40 mEq/L, but > 40 mEq/L the patient almost certainly has intrinsic renal functional impairment of chronic urinary tract obstruction.

The FENa has been found to be the most discriminating test. Virtually all patients with prerenal azotemia have an FENa < 1% whereas ATN patients usually have a value > 2%, although there may be some overlap in patients with nonoliguric ATN. The U/PUr and U/PCr ratios may also be useful, but again have a wide range of overlap. An U/PUr ratio < 3, however, is virtually never found in prerenal azotemia, whereas values > 8 are unlikely to be seen in ATN. Likewise, a U/PCr between 20 and 40 is associated with substantial overlap, but values below and above this range are quite helpful in differentiating ATN from prerenal azotemia.

## **REFERENCES (14)**

Stein JH: Current concepts in acute renal failure. Acute Care Therapeutics 1986;1:1.

Stein JH, Meyer DL, Barnes LD, et al. Current concepts in the pathophysiology of acute renal failure failure. Am J Physiol 1978; 243:F171.

Anderson RJ, Schrier RW. Clinical spectrum of liguric and nonoliguric acute renal failure. *In:* Brenner BM, Stein JH (Eds). *Acute Renal Failure*. New York, Churchill Livingstone, 1980:1.

Miller TJ, Anderson RJ, Linas SL, et al. Urinary diagnostic indices in acute renal failure: A prospective study. Ann Intern Med 1978;89:47.

Mathew A, Berl T. Fractional excretion of sodium: Use early to assess renal failure. *J Crit Illness* 1989;4:45.

Corwin HL, Bonventre JV. Acute renal failure in the intensive care unit (Parts I and II). Intensive Care Med 1988;14:10.

# SECTION 14: Surgery/Postoperative

# **SECTION 14: SURGERY/POSTOPERATIVE**

Instructions: For each	question listed	below, select	the most correct answer.
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A 45-year-old female is admitted to the surgical ICU after gastric bypass surgery. The
surgeon commented that it was difficult to close the abdomen. Overnight, the urine output
progressively decreased and was 30 mL over the last 4 hours. The intraabdominal drains have put
out a total of 140 mL. HR is 105/min, BP 110/50 mm Hg, central venous pressure 8 mm Hg,
and RR of 14/min. The abdomen appears more distended and tighter than on admission. The
intraabdominal pressure is 12 mm Hg, measured through the urinary catheter.

Which one of the following would you suggest?

- A. Fluid challenge
- B. Start dopamine at 2 µg/kg/min
- C. Diuresis
- D. Opening the abdominal incision
- E. Exchanging the urinary catheter
- 2. Which health benefits are attributed to bariatric surgery?
  - A. Resolution of sleep apnea
  - B. Control of type II diabetes
  - C. Improved management of hypertension
  - D. Decreased lipid levels
  - E. All of the above

3. A 32-year-old male developed a pseudocyst (8 cm) of the tail of the pancreas from alcohol-induced pancreatitis. He is in the postanaesthesia care unit after a distal pancreatectomy and splenectomy. He is pale, sweating, and complains of difficulty breathing and severe abdominal pain. His HR is 120/min, and his systolic BP is 98 mm Hg.

Which one of the following statements best describes his current clinical situation?

- A. The patient should be presumed to have intraabdominal bleeding and return to the operating room
- B. The patient should be treated for alcohol withdrawal
- C. The patient has pancreatitis-related shock with third space fluid sequestration
- D. The patient has sepsis and should be tested for culture data, as well as begin antibiotics
- E. A pulmonary artery catheter should be inserted to evaluate the patient's cardiac function
- 4. Which of the following is the best predictor of perioperative pulmonary complications?
  - A. Obesity
  - B. Controlled asthma
  - C. High Goldman Cardiac Risk Index
  - D. Age
  - E. Corticosteroid use
- 5. Which statement is least correct regarding physiologic changes in pulmonary function following surgery?
  - A. Physiologic changes are seen mainly with thoracic and upper abdominal procedures
  - B. Physiologic changes are decreased with smoking cessation 2 weeks prior to operation
  - C. Physiologic changes include diminished FEV,
  - D. Physiologic changes are independent of anesthetic choice
  - E. Duration of procedures does not affect physiologic changes

6. A 36-year-old man develops respiratory distress with stridor minutes after extubation following a difficult tonsillectomy. Chest radiograph excludes pneumothorax but shows diffuse infiltrates.

Which of the following is the optimal therapeutic response?

- A. Broad-spectrum antibiotics
- B. High-dose steroids
- C. Fiberoptic bronchoscopy
- D. Volume-control ventilation with positive end expiratory pressure
- E. Nebulized epinephrine
- 7. A 65-year-old male with chronic obstructive pulmonary disorder and insulin-dependent diabetes has just been extubated on postoperative day 4, following an emergency operation for an obstructing left-sided colon cancer. He is receiving nasal oxygen and bronchodilator therapy. Following an episode of coughing, his midline wound dressing is saturated with a large amount of salmon-colored fluid.

Which one of the following is the most appropriate intervention?

- A. Initiate antibiotics for a wound infection
- B. Observation for any further drainage
- C. A CT scan of the abdomen
- D. The patient will need to return to the operating room
- E. Open skin incision at the bedside
- 8. Which statement regarding perioperative cardiovascular risk modification is true?
  - A. A patient with good exercise tolerance (>4 METS) does not require further risk stratification for surgery
  - B. A patient with frequent premature ventricular contractions requires aggressive perioperative monitoring
  - C. Advanced age is a minor risk factor for perioperative cardiac complications
  - D. Uncontrolled systemic hypertension is an intermediate risk factor for perioperative cardiovascular events

9. An elderly male with chronic atrial fibrillation presents with an obstructing colon mass. Coronary artery bypass grafting was performed following myocardial infarction 3 years ago. The patient is not toxic, and peritonitis is absent. Nonetheless, the surgeon indicates that relief of large bowel obstruction is required.

Which of the following is indicated prior to surgery?

- A. Thallium stress test
- B. Emergency cardiac catheterization
- C. Pulmonary artery catheter placement for hemodynamic optimization
- D. Start β-blockade
- E. Start amiodarone

10. A 40-year-old female underwent uneventful laparoscopic cholecystectomy. Following the discharge from the hospital, she developed abdominal pain, vomiting, fever, and mental status changes. On examination, she has temperature of 39°C (102.2°F), HR 126/min, RR of 30/min, and systolic BP of 100 mm Hg. She has decreased breath sounds on the right and diffusely tender abdomen. She has minimal urine output.

Which one of the following is the most likely cause of the patient's symptoms?

- A. Pulmonary emboli
- B. Pneumonia
- C. Bile duct leak
- D. Bleeding
- E. Common bile duct obstruction

11. A 63-year-old patient with generalized peritonitis from perforated diverticulitis is admitted to the ICU after colon resection, colostomy, and Hartman pouch. Acute respiratory distress syndrome and renal failure requiring hemodialysis complicate the ICU course. During the second week of treatment, the patient develops a temperature of 39°C (102.2°F), abdominal distension, and intolerance of tube feedings.

Which one of the following is the most important diagnostic procedure?

- A. Abdominal radiographs
- B. Colonoscopy
- C. Blood cultures
- D. Examine the colostomy and perform a rectal exam
- E. Upright chest radiograph
- 12. A morbidly obese woman undergoes gastric bypass. Which is least likely to complicate her postoperative course in the first week?
  - A. Gastrointestinal leak
  - B. Stromal obstruction
  - C. Wound infection
  - D. Pulmonary embolism
  - E. Respiratory failure
- 13. An elderly female develops atrial fibrillation after cardiopulmonary bypass and coronary artery bypass grafting. Which statement is correct?
  - A. Digoxin is indicated for prophylaxis
  - B. Patients with chronic atrial fibrillation should return to heparin therapy as soon as possible
  - C. Ibutilide is used in patients with depressed ventricular function
  - D. Cardioversion is indicated in patients with depressed ventricular function

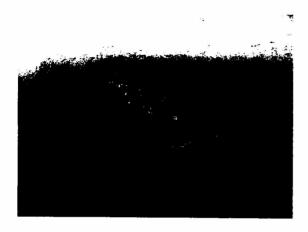
- 14. Which statement describing soft tissue infection and its therapy is *least* correct?
  - A. The most common surgical soft tissue infection is the subcutaneous abscess that resolves rapidly with incision and drainage
  - B. Cellulitis is adequately treated with antibiotics combined with local heat and elevation
  - C. Deep infections have a unique external appearance and debridement to the level of the fascia is required
  - D. Necrotizing fasciitis frequently is associated with minimal cutaneous change
- 15. & 16. A 68-year-old female is brought to the emergency department in shock after complaining of abdominal pain. A ruptured aortic aneurysm is diagnosed, and the patient is taken for emergent surgical repair. After a prolonged operation requiring massive amounts of crystalloids and blood products, the aneurysm is repaired. The patient is transferred to the ICU, intubated, and receiving vasopressor support. On arrival, her temperature is 36.6°C (97.8°F), BP 110/58 mm Hg, HR 92/min, and RR 20/min, with CMV 14, tidal volume 450 mL, positive end expiratory pressure +5 cm H<sub>2</sub>O and Fio<sub>2</sub> 100%. Urine output is 75 mL/h, patient continues to receive 0.9% NS at 100 mL/h. Laboratory data include the following: hemoglobin 9.4 g/dL, normal platelet count, normal electrolytes, and BUN/Cr of 25/1.6 mg/dL.

Ten hours after arrival to ICU, the BP drops to 80/36 mm Hg, and the central venous pressure is 18 mm Hg. Urine output has decreased to <50 mL/h over last 2 hours, despite increased IV fluids. Peak airway pressure increased from 20 cm H<sub>2</sub>O to 56 cm H<sub>2</sub>O, and the patient develops hypoxemia not responding to increased Fio<sub>2</sub> to 100% and PEEP of 12 cm H<sub>2</sub>O. Hemoglobin obtained from an arterial blood gas measurement is 9.2 g/dL.

- 15. Which one of the following diagnostic tests should be done first?
  - A. Transesophageal echocardiography
  - B. CT scan of abdomen and pelvis
  - C. Measurement of bladder pressure
  - D. Abdominal ultrasound
  - E. Placement of pulmonary artery catheter
- 16. Following the test above, the patient continues to deteriorate. Which one of the following interventions should be done next?
  - A. Aggressive resuscitation with packed red blood cells, fresh frozen plasma, and crystalloids
  - B. Emergent dialysis
  - C. Pericardiocentesis at the bedside
  - D. Emergent laparotomy
  - E. Change ventilator to pressure-control mode

17. A young male is admitted to ICU with acute alcohol intoxication. This open wound (see below) is seen on his abdominal wall. No drainage or fluctuance is appreciated.

Which of the following is the most appropriate management of this wound?



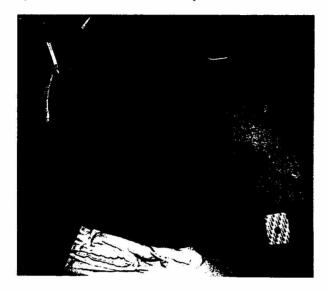
- A. Open the wound and pack
- B. Open the wound and initiate Gram-positive antibiotic coverage
- C. Initiate Gram-positive antibiotic coverage
- D. Leave the wound alone, and manage expectantly
- 18. A 63-year-old male is transferred to the surgical ICU 4 days after complicated, open, left hemicolectomy. His level of consciousness is decreased, and fever is present. There is new purulent drainage from his wound (shown below).



Which of the following is the most appropriate immediate therapy?

- A. Start fluid resuscitation and initiate Gram-positive antibiotic coverage
- B. Initiate Gram-negative aerobic and anaerobic antibiotic coverage with additional fluids
- C. Perform surgical exploration and initiate Gram-negative aerobic and anaerobic antibiotic coverage
- D. Takedown of colonic anastomosis with diverting colostomy

19. A middle-aged female presents with torso cellulitis, which has progressed over several days. She has fevers and chills. Past history is remarkable for an ipsilateral mastectomy with radiation and recurring local wound infections, which have been treated with oral antibiotics. The patient is transferred to the ICU with significant cellulitis, fevers, and multiorgan dysfunction. Her mastectomy incision is now healed, as pictured below.



Which one of the following therapeutic strategies is most appropriate?

- A. Provide vancomycin and IV fluids with later surgical intervention if the patient does not show rapid improvement
- B. Immediate surgical intervention with wound exploration and chest wall debridement, plus vancomycin and initiate Gram-negative antibiotic coverage
- C. Immediate administration of resuscitation fluids plus vancomycin
- D. Open the wound at the bedside, as the patient may become unstable in the operating room

20. A 65-year-old man has an 8-cm abdominal aortic aneurysm and back pain that subsides with BP control. Surgery is required in the next several days. Perioperative evaluation reveals a significant lesion in the right coronary artery.

Which of the following interventions is the most appropriate to reduce the risk of perioperative myocardial infarction?

- A. Percutaneous balloon angioplasty of the right coronary artery
- B. Percutaneous balloon angioplasty with metal stent placement in the right coronary artery
- C. Percutaneous balloon angioplasty with drug-eluting stent placement in the right coronary artery
- D. Off-pump coronary artery bypass grafting to the right coronary artery
- 21. A 49-year-old male undergoes a difficult cystectomy for cancer with extensive dissection and transfusion of 4 U of packed red blood cells. Postoperatively, he has 3 hours of hypotension in the surgical ICU, and troponin levels are elevated. Electrocardiograph indicates sinus tachycardia with no ST-segment changes.

Which of the following therapies is least appropriate?

- A. Aggressive pain relief
- B. β-Blockade
- C. Low-molecular-weight heparin
- D. IV fluids
- 22. A 42-year-old female undergoes laparoscopic cholecystectomy. Twelve hours after surgery, the patient has tachypnea and obvious cervical crepitus. Respiratory rate is 20/min with Sao<sub>2</sub> 95% while receiving room air. Chest radiograph reveals cervical subcutaneous emphysema and no evidence of pneumothorax.

Which of the following treatment options is least appropriate?

- A. Immediate thoracotomy
- B. Intubation with placement of the endotracheal tube just above the carina
- C. Continued spontaneous breathing, as tolerated
- D. Noninvasive ventilation
- E. Bilateral tube thoracostomy

- 23. Which one of the following statements about abdominal pain is true?
  - A. A rigid abdomen reflects irritation of the visceral peritoneum
  - B. Perforated peptic ulcer disease is typically manifest as a gradually progressing upper abdominal discomfort
  - C. Right lower quadrant pain in appendicitis reflects parietal peritoneal irritation
  - D. CT scan is the diagnostic test of choice with generalized peritonitis
  - E. Hemorrhage into the peritoneal cavity will cause diffuse abdominal pain
- 24. A 50-year-old male undergoes laparoscopic gastric bypass. Postoperatively, he complains of numbness and muscular pain in his lower back and buttocks. Purpura is seen at these sites. In the past 4 hours, a small amount of urine with increased pigment load is seen. Erythrocytes are absent on urinalysis.

Which statement regarding this problem is most correct?

- A. Urgent surgical gluteal compartment decompression is required
- B. Treatment is initiated at a creatine phosphokinase level of 10,000 U/L
- C. Acute renal failure occurs in 50% of patients
- D. Recovery of renal function is unlikely
- 25. Which one of the following patients has the highest risk for morbidity and mortality following surgery?

Patient	Age	Comorbidity*	Exercise Tolerance	Surgery	Anesthetic
A.	58	Hypertension	Good	Emergent for ruptured AAA	General
B.	90	CAD, COPD	Poor	Elective cataract removal	Local
C.	75	None	Moderate	Elective colectomy	General
D.	80	CAD, HTN	Moderate	Emergent hip fracture repair	Epidural

<sup>\*</sup> CAD, coronary artery disease; COPD, chronic obstructive pulmonary disorder; HTN, hypertension

# SECTION 14: SURGERY/POSTOPERATIVE

#### **ANSWERS:**

1-A; 2-E; 3-A; 4-C; 5-E; 6-D; 7-D; 8-C; 9-D; 10-C; 11-D; 12-C; 13-B; 14-D; 15-C; 16-D; 17-D; 18-C; 19-B; 20-A; 21-C; 22-E; 23-C; 24-C; 25-A

RATIONALE (1)

Answer: A

Normal intraabdominal pressure (IAP) is approximately 5 mm Hg. Increased IAP occurs in 4 to 15% of surgical ICU settings in patients in a variety of clinical situations, including lengthy abdominal surgery, ascites accumulation, blunt abdominal trauma, ruptured abdominal aortic aneurysm, hemorrhagic pancreatitis, pelvic fractures, ileus, intestinal obstruction, and pneumoperitoneum. In addition to intraabdominal processes, external factors, such as large pannus or abdominal binder, can cause an increase in IAP.

Abdominal compartment syndrome is characterized by an increased IAP, increased peak inspiratory pressure, decreased urinary output, and decreased venous return with hypotension. Measurements from a variety of catheters, including transvesical, transgastric, rectal, and femoral venous cannulas, have been used to reflect IAP. Currently, measuring IAP through a urinary catheter is the most popular method. An IAP of more than 12 mm Hg is considered abnormal. However, it is unclear what length of time intraabdominal hypertension can be tolerated before significant end-organ damage occurs. In the absence of good outcome data, expert consensus is that increases of IAP ≥20 mm Hg with end-organ compromise warrant urgent decompression. After abdominal decompression, signs and symptoms of compartment syndrome rapidly resolve, but patients may experience fluid shifts and hemodynamic instability.

Renal dysfunction accompanying elevations in IAP is characterized by oliguria unresponsive to volume expansion. Oliguria can be seen when IAPs reach 15 mm Hg, with anuria at pressures greater than 30 mm Hg. The mechanism of renal dysfunction may involve pressure related decrease in renal arterial flow leading to decreasing glomerular filtration rate. Pressure-related ureteral obstruction is not thought to contribute to renal failure. Low-dose dopamine is not used to enhance renal performance. There is no evidence of low cardiac output here.

In this case, giving additional fluids first makes the most sense, because the patient has no other sign of acute abdominal compartment syndrome, the Foley catheter appears to be patent because of continued urine output, and the central venous pressure is not elevated. Also, the patient did not receive an excessive amount of fluid for this case, and significant amounts of fluid can shift from the intravascular to the interstitial and intraperitoneal space during the first day or 2 after surgery. There is no indication for afterload reduction. A diuretic should not be used until it is confirmed that there is adequate intravascular volume.

#### **REFERENCES (1)**

- Cheatham ML, White MW, Sagraves SG, et al. Abdominal perfusion pressure: a superior parameter in the assessment of intra-abdominal hypertension. *J Trauma* 2000; 49:621-627.
- Ertel W, Oberholzer A, Platz A, et al. Incidence and clinical pattern of the abdominal compartment syndrome after "damage-control" laparotomy in 311 patients with severe abdominal and/or pelvic trauma. *Crit Care Med* 2000; 28:1747-1753.
- Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Results from the International Conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. I. Definitions. *Intensive Care Med* 2006;32:1722-1732.
- McNelis J, Marini CP, Jurkiewicz A, et al. Predictive factors associated with the development of abdominal compartment syndrome in the surgical intensive care unit. *Arch Surg* 2002; 137:133-136.
- World Society of the Abdominal Compartment Syndrome. Preliminary consensus definitions on intraabdominal hypertension (IAH) and abdominal compartment syndrome (ACS): results from the International ACS Consensus Definitions Conference. Available at: www.wsacs.org. Accessed June 12, 2007.

RATIONALE (2)

Answer: E

While bariatric surgery does not eliminate obesity, the metabolic advantages are remarkable. Strong evidence for improvement in type II diabetes and impaired glucose tolerance was found across all surgery types. Hyperlipidemia, hypercholesterolemia, and hypertriglyceridemia are also significantly improved across all surgical procedures. Hypertension is also better managed after bariatric surgery. Perhaps most remarkable is improved outcome of obstructive sleep apnea in patients carrying a diagnosis of sleep apnea, sleep-disordered breathing, and Pickwickian syndrome. In a large, recent metaanalysis, the percentage of patients in the total population whose obstructive sleep apnea resolved was over 85%.

### **REFERENCES (2)**

- Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. *JAMA* 2004; 292:1724-1737.
- Pories WJ, Swanson MS, MacDonald KG, et al. Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes mellitus. *Ann Surg* 1995; 222:339-352.
- Rasheid S, Banasiak M, Gallagher SF, et al. Gastric bypass is an effective treatment for obstructive sleep apnea in patients with clinically significant obesity. *Obes Surg* 2003; 13:58-61.
- Sugerman HJ, Wolfe LG, Sica DA, et al. Diabetes and hypertension in severe obesity and effects of gastric bypass-induced weight loss. *Ann Surg* 2003; 237:751-758.

RATIONALE (3)

Answer: A

Physicians are more attuned to postoperative bleeding in trauma operations than in elective operations. Pancreatitis creates intense inflammation around the area of the pancreas, and resection of a pseudocyst occurs in this area of inflammation. A pseudocyst of the tail of the pancreas usually involves the spleen. The spleen has both short gastric arteries and the major splenic artery and veins that must be ligated during removal of the tail of the pancreas and spleen. In the face of inflammation, unnamed vessels may be present in the other ligaments, which attach to the spleen. Prior to closure, absolute hemostasis is achieved. However, delayed bleeding can occur from technical errors or gastric distension. The fluid from drainage tubes can be misleading.

Hypovolemic shock should be suspected in the patient described above, and a return to the operating room is required to control bleeding. Severe ethyl alcohol withdrawal usually presents later in the hospital course and is associated with sweating, tachycardia, and hypertension. Abdominal pain is not a component of severe ethyl alcohol withdrawal. Epidural pain control would increase the degree of hypotension. Sepsis is unlikely, unless the patient had a preoperative infection. Acute-postoperative-pancreatitis-induced shock would not be expected. There is no indication for a pulmonary artery catheter. The patient needs fluid resuscitation and reexploration.

#### **REFERENCES (3)**

Cooperman AM. Surgical treatment of pancreatic pseudocysts. Surg Clin North Am 2001; 81:411-419. Dayton MT. Surgical complications. In: Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 17th ed. Philadelphia, PA: Elsevier Saunders; 2004:297-332. Tsuei BJ, Schwartz RW. Current management of pancreatic pseudocysts. Curr Surg 2003; 60:587-590.

RATIONALE (4)

Answer: C

A number of patient-related factors have been repeatedly shown to increase the likelihood of pulmonary morbidity associated with surgical procedures. Chronic obstructive pulmonary disorder, smoking, and poor general health status are accepted predictive factors. Poor general health status can be predicted by ASA class >2 or an elevated Goldman Cardiac Risk Index. There are conflicting data regarding age. Increased perioperative morbidity associated with age may relate to comorbid conditions. Obesity does not appear to be a risk factor and reactive airway disease (asthma) should not increase operative risk if asthma is well controlled during the preoperative period.

#### **REFERENCES (4)**

- Abir F, Bell R. Assessment and management of the obese patient. Crit Care Med 2004;32(Suppl): S87-S91.
- Ali MR, Maguire MB, Wolfe BM. Assessment of obesity-related comorbidities: A novel scheme for evaluating bariatric surgical patients. *J Am Coll Surg* 2006;202:70-77.
- Arozullah AM, Conde MV, Lawrence VA. Preoperative evaluation for postoperative pulmonary complications. *Med Clin North Am* 2003; 87:153-173.
- Dales RE, Dionne G, Leech JA, et al. Preoperative prediction of pulmonary complications following thoracic surgery. *Chest* 1993; 104:155-159.
- Pasulka PS, Bistrian BR, Benotti PN, et al. The risks of surgery in obese patients. *Ann Intern Med* 1986; 104:540-546.
- Warner DO, Warner MA, Barnes RD, et al. Perioperative respiratory complications in patients with asthma. *Anesthesiology* 1996; 85:460-467.

RATIONALE (5)

Answer: E

A variety of postoperative pulmonary changes have been described after surgery and anesthesia. Decreased vital capacity, FRC, FEV<sub>1</sub> and tidal volume have been reported. These changes can occur with a variety of operative procedures but are most prominent with thoracic and upper abdominal surgery. Diaphragmatic dysfunction associated with pain and splinting has been associated particularly with surgical procedures adjacent to the diaphragm. When FRC is less than closing volume, atelectasis with ventilation perfusion mismatch is likely. A change in ventilatory response is noted after some anesthetic agents with compromise of cough and mucocilliary clearance. There is a clear relationship to duration of operative procedure and anesthetic choice. Neuroaxial blockade carries fewer pulmonary risk factors than general anesthesia. Acute dysfunction with smoking takes several weeks to subside and increases in proportion to preoperative amount of smoking.

## REFERENCES (5)

Rodgers A, Walker N, Schug S, et al. Reduction of postoperative mortality and morbidity with epidural or spinal anaesthesia: Results from overview of randomised trials. BMJ 2000; 321:1493.
Smetana GW. Preoperative pulmonary evaluation. N Engl J Med 1999; 340:937-944.
Smetana GW, Cohn SL, Lawrence VA. Update in preoperative medicine. Ann Intern Med 2004; 140:452-461.

RATIONALE (6)

Answer: D

This patient has negative pressure pulmonary edema from forced inspiration against a closed glottis consistent with upper airway obstruction. Edema frequently develops after obstruction is acutely relieved. In general, edema appears immediately but may appear over subsequent hours. Supportive treatment with mechanical ventilation is the most appropriate intervention.

The overall prevalence of aspiration is relatively low. Large series document <1% incidence in this setting. Risk factors for aspiration include non-white males, age >60 years, dementia, chronic obstructive pulmonary disorder, renal disease, malignancy, hepatic disease, and emergency procedures. Overall, mortality is low, with aspiration following operative procedures. If significant pulmonary dysfunction is not seen within the initial 2 hours, no sequelae are likely. There is no role for prophylactic antibiotics or steroids.

#### **REFERENCES (6)**

Arieff AI. Fatal postoperative pulmonary edema: Pathogenesis and literature review. *Chest* 1999; 115:1371-1377.

Timby J, Reed C, Zeilender S, et al. "Mechanical" causes of pulmonary edema. *Chest* 1990; 98:973-979.

Warner MA, Warner ME, Weber JG. Clinical significance of pulmonary aspiration during the perioperative period. *Anesthesiology* 1993; 78:56-62.

RATIONALE (7)

Answer: D

This patient has wound dehiscence, which typically occurs on postoperative day 4 or 5. Wound dehiscence is characterized by profuse drainage of salmon-colored fluid that soaks the dressing and bedding. Wound dehiscence is a technical problem with the wound closure technique allowing the fascia to open. It can lead to evisceration of abdominal contents. The dressing should be removed to examine the wound. If bowel is exposed, wet saline packs are placed, and the patient returns to the operating room for wound closure. There is a role for nonoperative management with moist dressings and an abdominal binder. Risk factors for wound dehiscence include age >45 years, malnutrition, morbid obesity, cancer, uremia, diabetes, coughing, and other causes of increased intraabdominal pressure, infection, and hemorrhage. Unless an intraabdominal abscess is suspected, a CT scan of the abdomen is not indicated. Long-term outcome issues associated with wound dehiscence include incisional hernias.

#### **REFERENCES (7)**

Page CP, Bohnen JM, Fletcher JR, et al. Antimicrobial prophylaxis for surgical wounds: guidelines for clinical care. *Arch Surg* 1993; 128:79-88.

Richards PC, Balch CM, Aldrete JS. Abdominal wound closure: a randomized prospective study of 571 patients comparing continuous vs interrupted suture techniques. *Ann Surg* 1983; 197:238-243. Riou JP, Cohen JR, Johnson H Jr. Factors influencing wound dehiscence. *Am J Surg* 1992; 163:324-330.

RATIONALE (8)

Answer: C

Noncardiac surgery is generally safe for patients with moderate or excellent exercise capacity (4 METS or greater) and who lack intermediate or major predictors of clinical risk. Other individuals should be considered for additional testing. Frequent ventricular premature beats and asymptomatic nonsustained ventricular tachycardia have not been associated with an increased risk of nonfatal myocardial infarction or cardiac death during the perioperative period. Aggressive monitoring in the perioperative period is not necessary.

The American College of Cardiology and American Heart Association have developed guidelines on perioperative cardiovascular evaluation for patients undergoing noncardiac surgery. Major predictors of increased perioperative cardiovascular risk include unstable coronary syndromes, decompensated heart failure, symptomatic ventricular arrhythmias, or high-grade atrioventricular ventricular block and severe valvular disease. Intermediate risk is marked by renal insufficiency, diabetes mellitus, heart failure, myocardial infarction by history and mild angina pectoris. Minor predictors include advanced age, electrocardiograph abnormality, atrial fibrillation, reduced functional capacity, history of stroke, and uncontrolled systemic hypertension.

#### **REFERENCES (8)**

- Akhtar S, Silverman DG. Assessment and management of patients with ischemic heart disease. *Crit Care Med* 2004;32(Suppl):S126-S136.
- Eagle KA, Berger PB, Calkins H, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery [executive summary]. *Circulation* 2002; 105:1257-1267.
- Fletcher GF, Balady G, Froelicher VF, et al. Exercise standards. a statement for healthcare professionals from the American Heart Association. *Circulation* 1995; 91:580-615.
- Mahla E, Rotman B, Rehak P, et al. Perioperative ventricular dysrhythmias in patients with structural heart disease undergoing noncardiac surgery. *Anesth Analg* 1998; 86:16-21.
- O'Kelly B, Browner WS, Massie B, et al. Ventricular arrhythmias in patients undergoing noncardiac surgery: the Study of Perioperative Ischemia Research Group. *JAMA* 1992; 268:217-221.

RATIONALE (9)

Answer: D

This patient has an obstructing colon mass, presumably neoplastic. There is no history of new coronary symptoms. If the patient has a stable clinical status from the standpoint of coronary artery disease, no additional cardiac testing is required for coronary artery bypass grafting occurring within the previous 5 years. There are no consistent data supporting the use of pulmonary artery catheters in the perioperative period. Placement of a pulmonary artery catheter may be considered when significant fluid shifts are anticipated. Hypertension with systolic BP  $\geq$ 180 mm Hg and diastolic BP  $\geq$ 110 mm Hg should be controlled before surgery. Rapid acting agents can be administered to allow effective control in a matter of hours.  $\beta$ -blockers are particularly attractive agents. Amiodarone does not have cardioprotective effects in the patient undergoing noncardiac surgery.

#### **REFERENCES (9)**

- Devereaux PJ, Beattie WS, Choi PTL, et al. How strong is the evidence for the use of perioperative β-blockers in non-cardiac surgery? Systematic review and metaanalysis of randomised controlled trials. *BMJ* 2005; 331:313-321.
- Eagle KA, Berger PB, Calkins H, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery: executive summary a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation* 2002; 105:1257-1267.
- Polanczyk CA, Rohde LE, Goldman L, et al. Right heart catheterization and cardiac complications in patients undergoing noncardiac surgery: an observational study. *JAMA* 2001; 286:309-314.
- Practice guidelines for pulmonary artery catheterization. A report by the American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. *Anesthesiology* 1993; 78:380-394.
- Sandham JD, Hull RD, Brant RF, et al. A randomized, controlled trial of the use of pulmonary-artery catheters in high-risk surgical patients. N Engl J Med 2003; 348:5-14.

RATIONALE (10)

Answer: C

This patient has symptoms and signs of sepsis after her laparoscopic cholecystectomy. The most common causes of sepsis are bowel perforation secondary to trocar placement, burn injury to the bowel from electocautery, and a bile duct leak from the cystic duct. Cystic duct leaks represent one half of the bile duct injuries after laparoscopic cholecystectomy (0.5%). More severe forms of duct injury are associated with diminished survival after cholecystectomy. Cystic duct leaks typically occur, because the clip comes loose or there are other accessory bile ducts draining from the liver. The intraperitoneal bile causes peritonitis. A cystic duct leak can usually be managed with endoscopic stenting and percutaneous drainage. Occasionally, reoperation is required.

This patient will require fluid resuscitation. A bedside right upper-quadrant ultrasound can confirm a collection of fluid below the liver that can be aspirated and a drain placed. If bile is obtained, an endoscopic retrograde cholangiopancreatography can confirm the anatomic position of the bile leak and a stent can be placed for internal drainage. A CT scan, with percutaneous drainage, can also be performed. If fluid aspiration shows bowel contents, laporatomy is planned. Pulmonary embolus can occur during laparoscopic cholecystectomy but does not present with abdominal pain. Pneumonia can cause sepsis, but the abdominal pain is localized to the upper abdomen. Bleeding is not usually associated with high temperatures. Common duct obstruction usually presents with jaundice.

#### **REFERENCES (10)**

- Branum G, Schmitt C, Baillie J, et al. Management of major biliary complications after laparoscopic cholecystectomy. *Ann Surg* 1993; 217:532-541.
- Flum DR, Cheadle A, Prela C, et al. Bile duct injury during cholecystectomy and survival in medicare beneficiaries. *JAMA* 2003; 290:2168-2173.
- Giger UF, Michel JM, Opitz I, et al. Risk factors for perioperative complications in patients undergoing laparoscopic cholecystectomy: Analysis of 22,953 consecutive cases from the Swiss Association of Laparoscopic and Thoracoscopic Surgery database. *J Am Coll Surg* 2006;203:723-728.
- de Reuver PR, Grossmann I, Busch OR, et al. Referral pattern and timing of repair are risk factors for complications after reconstructive surgery for bile duct injury. *Ann Surg* 2007;245:763-770.
- Martin RF, Rossi RL. Bile duct injuries: spectrum, mechanisms of injury, and their prevention. Surg Clin North Am 1994; 74:781-807.
- McLean TR. Risk management observations from litigation involving laparoscopic cholecystectomy. Arch Surg 2006;141:643-648.
- Moore DE, Feurer ID, Holzman MD, et al. Long-term detrimental effect of bile duct injury on health-related quality of life. Arch Surg 2004;139:476-482.

RATIONALE (11)

Answer: D

In a postoperative patient, fever >39°C (102.2°F) after the fifth postoperative day suggests wound infection, anastomotic breakdown, or an intraabdominal abscess/pelvic abscess. Ileus presenting as intolerance to tube feeding, abdominal distension, or absent air or stool in the ostomy bag occurs frequently with an intraabdominal abscess. If the ostomy is blue, purple, or black, colonic ischemia should be suspected. Digital examination of the ostomy can be used to evaluate fecal impaction or a tight fascial ostomy site. A rectal examination with anterior bogginess or bulge is suspicious for a pelvic abscess. One third of all abscesses occur after episodes of peritonitis. Abdominal and chest radiographs should be performed. Radiographs can be obtained after your examination. The chest radiograph is examined for free air or new infiltrates. The abdominal radiograph is examined for ileus, obstruction, or signs of an abscess. Postoperative pancreatitis usually occurs in the early postoperative period and is unlikely this late. Culture data should be obtained from obvious sites and antibiotics started. All these are done after the patient is examined.

## **REFERENCES (11)**

- Anaya DA, Dellinger EP. Surgical infections and choice of antibiotics. In: Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 17th ed. Philadelphia, PA: Elsevier Saunders; 2004:257-282.
- Dellinger E: Approach to the patient with postoperative fever. In: Gorbach S, Bartlett J, Blacklow N, eds. *Infectious Diseases in Medicine and Surgery*. Philadelphia, PA: WB Saunders; 1998:903-909.
- Parcelli F, Doglietto GB, Alfieri S, et al. Prognosis in intraabdominal infections: Multivariate analysis on 604 patients. Arch Surg 1996; 131:641-645.

RATIONALE (12)

Answer: C

The major complications of bariatric surgery include pulmonary embolism, respiratory failure, gastrointestinal leaks from the breakdown of a staple or suture line, stromal obstruction, or stenosis, and bleeding. A wound infection is less likely to be a complication in the first week. The level of any complication risk is related to specific procedure and patient age, degree of obesity, and associated medical conditions. Postoperative mortality is thought to range from as low as 0.1% to as high as 1 to 2%.

#### **REFERENCES (12)**

Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and metaanalysis. *JAMA* 2004; 292:1724-1737.

Ceelen W, Walder J, Cardon A, et al. Surgical treatment of severe obesity with a low-pressure adjustable gastric band: Experimental data and clinical results in 625 patients. *Ann Surg* 2003; 237:10-16.

DeMaria EJ. Bariatric surgery for morbid obesity. N Engl J Med 2007;356:2176-2183.

El-Solh AA. Clinical approach to the critically ill, morbidly obese patient. Am J Respir Crit Care Med 2004;169:557-561.

Kalfarentzos F, Dimakopoulos A, Kehagias I, et al. Vertical banded gastroplasty versus standard or distal Roux-en-Y gastric bypass based on specific selection criteria in the morbidly obese: preliminary results. *Obes Surg* 1999; 9:433-442.

Pieracci FM, Barie PS, Pomp A. Critical care of the bariatric patient. *Crit Care Med* 2006;34:1796-1804. Steinbrook R. Surgery for severe obesity. *N Engl J Med* 2004; 350:1075-1079.

Sugerman HJ, Wolfe LG, Sica DA, et al. Diabetes and hypertension in severe obesity and effects of gastric bypass-induced weight loss. *Ann Surg* 2003; 237:751-758.

RATIONALE (13)

Answer: B

The weight of data supports prophylaxis against atrial fibrillation in patients undergoing cardiac surgery. While the role of acute anticoagulation must be tempered with bleeding risks following surgery, it is clear that patients with chronic atrial fibrillation should return to anticoagulation therapy as soon as possible. Initially, this therapy may come from heparin administration with transition to coumadin.

The patient with new-onset atrial fibrillation and depressed ventricular function is best treated with amiodarone. A variety of agents are available for patients with good ventricular function including sotalol, ibutilide, amiodarone, procainamide, quinidine, and disopyramide. Immediate cardioversion is essential only for patients with acute hemodynamic instability typically reflected by hypotension (systolic BP <90 mm Hg).

Duration of antiarrhythmic therapy for patients suffering atrial fibrillation following cardiac surgery remains unclear. Thirty days of therapy is the current consensus recommendation, due to the tendency of patients suffering this problem to experience recurrence in the initial weeks after operation.

#### **REFERENCES (13)**

- American College of Chest Physicians guidelines for the prevention and management of postoperative atrial fibrillation after cardiac surgery. *Chest* 2005; 128:1S-64S.
- Crystal E, Connolly SJ, Sleik K, et al. Interventions on prevention of postoperative atrial fibrillation in patients undergoing heart surgery: a meta-analysis. *Circulation* 2002; 106:75-80.
- Roy D, Talajic M, Dorian P, et al. Amiodarone to prevent recurrences of atrial fibrillation: Canadian Trial of Atrial Fibrillation. *N Engl J Med* 2000; 342:913-921.
- St. Andre AC, DelRossi A. Hemodynamic management of patients in the first 24 hours after cardiac surgery. Crit Care Med 2005; 33:2082-2093.

RATIONALE (14)

Answer: D

Subcutaneous abscess is the most common soft tissue infection, which may involve a surgeon. If incision and drainage are effective, adjunctive antibiotics are indicated only for patients with chronic disease associated with immunosuppression or poorly localized infection.

Most soft tissue infections do not require a surgeon. Intradermal infections are primarily folliculitis (pyogenic organisms) or cellulitis (nonpyogenic organisms). Acute superficial abscess in the subcutaneous tissue is frequently drained without antibiotic therapy. However, chronic abscesses may contain necrotic tissue or spread to deeper levels and cause multiple draining sinuses of a carbuncle. Excision of necrotic tissue usually to the fascia level with delayed skin grafting may be required. Cellulitis may also spread in dermal lymphatics causing classical lymphangitis commonly caused by *Streptococcus pyogenes*.

Deep infections may be indistinguishable from cellulitis or have minimal cutaneous signs. Signs of systemic inflammation and remote organ dysfunction suggest involvement of infecting organisms deep to the skin at the level of fascial plane or below.

Simple cellulitis is adequately managed with antibiotics, heat, and elevation.

#### **REFERENCES (14)**

Ahrenholz DH. Necrotizing fasciitis and other soft tissue infections. In: Irwin RS, Rippe JM, eds. *Irwin and Rippe's Intensive Care Medicine*. Philadelphia, PA: Lippincott, Williams & Wilkins; 2003:1709-1716.

Bosshardt TL, Henderson VJ, Organ CH Jr. Necrotizing soft-tissue infections. *Arch Surg* 1996; 131:846-854.

Swartz MN. Clinical practice. Cellulitis. N Engl J Med 2004; 350:904-912.

RATIONALE (15)

Answer: C

This patient has developed hypotension, high airway pressures with hypoxemia, and oliguria. These findings are consistent with abdominal compartment syndrome (ACS). Commonly described in patients postlaparotomy for blunt and penetrating abdominal trauma, ACS was initially described after repair of ruptured abdominal aortic aneurysm. Other conditions associated with ACS are retroperitoneal hemorrhage, burns, peritonitis, liver transplantation, pancreatitis, and ascites. Persistent increase in intraabdominal pressure will produce multiple organ dysfunction. Hypotension results from a combination of decreased venous return and increased intrathoracic pressure (decreases left ventricular compliance). Respiratory failure with increased peak ventilatory pressures, hypoxemia, and eventual hypercarbia is seen with increased intraabdominal pressures. As intraabdominal pressure increases, a progression from oliguria to anuria is commonly seen. Documentation of an increased intraabdominal pressure is key in establishing the correct diagnosis and directing therapy.

Currently, urinary bladder pressure measurement is the most simple, reliable, and accepted methodology. A Foley catheter attached to a water manometer can be used. Intraabdominal hypertension is seen with intraabdominal pressures ≥12 mm Hg. ACS warranting decompressive laparotomy is defined by intraabdominal pressure of ≥20 mm Hg in association with new onset single or multiple organ failure. However, the decision to decompress the abdomen may come at pressures less than 20 mm Hg, if intraabdominal hypertension is seen with organ failure related to this problem.

Decisions for surgical decompression should take into account the clinical findings and the actual pressure. A transesophageal echocardiography would be of no value in this case, because there is no evidence of cardiac or thoracic aorta pathology causing clinical findings. A CT scan or abdominal ultrasound would not reveal further information. A new collection of blood, detectable by these imaging tests, is unlikely considering hemoglobin has remained stable. Pulmonary artery catheter pressures will not aid in making a correct diagnosis.

#### **REFERENCES (15)**

- Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Results from the International Conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. I. Definitions. *Intensive Care Med* 2006;32:1722-1732.
- Schein M, Wittmann DH, Aprahamian CC, et al. The abdominal compartment syndrome: the physiological and clinical consequences of elevated intraabdominal pressure. *J Am Coll Surg* 1995; 180:745-753.
- World Society of the Abdominal Compartment Syndrome. Preliminary consensus definitions on intraabdominal hypertension (IAH) and abdominal compartment syndrome (ACS): results from the International ACS Consensus Definitions Conference. Available at: www.wsacs.org. Accessed June 12, 2007.

RATIONALE (16)

Answer: D

In this patient with significant organ dysfunction from abdominal compartment syndrome immediate relief of increased abdominal pressure is mandatory. Of the options provided, a bedside laparotomy is the only treatment that will achieve this goal. The patient is critically ill and even delaying abdominal decompression to transport the patient to an operating room might be deleterious. Abdominal decompression will dramatically improve peak ventilatory pressures, hypoxemia, hypotension, and oliguria in this patient. There is no indication of acute hemorrhage requiring transfusion of blood products. Although fluid resuscitation can be used to treat increased intraabdominal pressure in early abdominal compartment syndrome, it will not be effective in patients with severe organ dysfunction. Emergent dialysis is not indicated for this patient since the oliguria is secondary to increased intraabdominal pressure. A pericardiocentesis is not indicated since there is no evidence of cardiac tamponade. Finally, changing the ventilator mode to pressure-control might decrease peak airway pressure but will not result in prolonged improvement in oxygenation.

#### **REFERENCES (16)**

- Balogh Z, McKinley BA, Holcomb JB, et al. Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. *J Trauma* 2003; 54:848-861.
- Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Results from the International Conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. I. Definitions. *Intensive Care Med* 2006;32:1722-1732.
- McNelis J, Marini CP, Jurkiewicz A, et al. Predictive factors associated with the development of abdominal compartment syndrome in the surgical intensive care unit. Arch Surg 2002; 137:133-136.
- Meldrum DR, Moore FA, Moore EE, et al. Prospective characterization and selective management of the abdominal compartment syndrome. Am J Surg 1997; 174:667-673.
- Morken J, West MA. Abdominal compartment syndrome in the intensive care unit. Curr Opin Crit Care 2001; 7:268-274.
- Orlando R 3rd, Eddy VA, Jacobs LM Jr., et al. The abdominal compartment syndrome. *Arch Surg* 2004; 139:415-422.

RATIONALE (17)

Answer: D

This patient has minimal cutaneous erythema and no clinical signs of toxicity. This local cutaneous reaction frequently reflects excessive granulation tissue formation in a wound allowed to close without suturing. The visible gap may reflect skin approximation under tension. Removal of any offending foreign body or necrotic tissue is adequate treatment, and the patient may be followed expectantly. Antibiotic therapy is inappropriate.

#### REFERENCES (17)

Ahrenholz DH. Necrotizing fasciitis and other soft tissue infections. In: Irwin RS, Rippe JM, eds. *Irwin and Rippe's Intensive Care Medicine*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003:1709-1716.

Anaya DA, Dellinger EP. Surgical infection and choice of antibiotics. In: Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery: The Biological Basis of Modern Surgical Practice. Philadelphia, PA: Elsevier Saunders; 2004:257-282.

RATIONALE (18)

Answer: C

This patient has a surgical site infection with indications for operative exploration. In this situation, infection involves the abdominal wall, and no evidence of anastomotic disruption is given.

Organisms are those found in the colon, including anaerobes and Gram-negative data.

In this setting, the diagnosis of a deep soft tissue infection involving the fascia requires a high index of suspicion. Initial findings are localized pain and swelling, often with no visible trauma or discoloration of the skin. Deep tissue sites, especially surgical wounds in perirectal areas, have the fewest signs and symptoms. As the infection spreads along fascial planes, dermal induration and erythema may become prominent. Bacteremia is rare. The diagnosis is confirmed when a probe or examiner's finger can be passed laterally along the fascial cleft when the wound is opened in the operating room.

Sverdrup and coworkers differentiate superficial and deep Streptococcal infection by systemic signs. Signs of deep infection included confusion, shock, marked local pain, pitting edema, bullae, cyanosis, or skin necrosis.

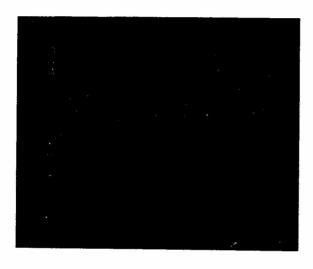
#### **REFERENCES (18)**

- Brandt MM, Corpron CA, Wahl WL. Necrotizing soft tissue infections: a surgical disease. *Am Surg* 2000; 66:967-971.
- Christou NV, Nohr CW, Meakins JL. Assessing operative site infection in surgical patients. *Arch Surg* 1987; 122:165-169.
- Mangram AJ, Horan TC, Pearson ML, et al. Guideline for prevention of surgical site infection, 1999. Infect Control Hosp Epidemiol 1999; 20:250-278.
- Sverdrup B, Blomback M, Borglund E, et al. Blood coagulation and fibrinolytic systems in patients with erysipelas and necrotizing fasciitis. *Scand J Infect Dis* 1981; 13:29-36.
- Weiss CA 3rd, Statz CL, Dahms RA, et al. Six years of surgical wound infection surveillance at a tertiary care center: review of the microbiologic and epidemiological aspects of 20,007 wounds. *Arch Surg* 1999; 134:1041-1048.

RATIONALE (19)

Answer: B

This patient has several indications for immediate operation. High fever, local immunocompromise secondary to irradiation, and an operative site with repeated soft tissue infections all indicate that careful evaluation of the chest wall with soft tissue debridement are indicated. Antibiotic administration alone or local wound exploration will not be adequate in this patient. Her old operative site (see below) was filled with fluid and fibrinous debris.



#### **REFERENCES (19)**

Ahrenholz DH. Necrotizing fasciitis and other soft tissue infections. In: Irwin RS, Rippe JM, eds. *Irwin and Rippe's Intensive Care Medicine*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003:1709-1716.

Bilton BD, Zibari GB, McMillan RW, et al. Aggressive surgical management of necrotizing fasciitis serves to decrease mortality: a retrospective study. *Am Surg* 1998; 64:397-401.

Dayton MT. Surgical complications. In: Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery: The Biological Basis of Modern Surgical Practice. 17th ed. Philadelphia, PA: Elsevier Saunders; 2004:297-332.

Swartz MN. Cellulitis. N Engl J Med 2004; 350:904-912.

RATIONALE (20)

Answer: A

Major thoracoabdominal procedures, as well as head and neck operations, are associated with high risk of cardiovascular complications. While emergency procedures, in general, are associated with higher risk, patients undergoing vascular surgical procedures are at *highest* risk for perioperative myocardial infarction. In part, this is because risk factors for coronary artery disease and vascular disease overlap, and these individuals have a higher prevalence of asymptomatic coronary disease.

With the increase in percutaneous coronary revascularization, the role of balloon angioplasty and stent implantation has been investigated. Patients who have recently undergone coronary revascularization with percutaneous techniques and stent implantation may be at *increased* risk of perioperative stent thrombosis and myocardial infarction. The American Heart Association recommends delay of 4 to 6 weeks between implantation of a bare metal stent and noncardiac surgery to reduce the risk of perioperative myocardial infarction. This allows 4 weeks of antiplatelet therapy during stent reendothelialization. The risk of stent thrombosis may be further extended in patients implanted with a drug-eluting stent, as these drugs prevent neointimal proliferation with delays in stent endothelialization. A recent review recommends delaying elective surgery for 3 months following a sirolimus-eluting stent and 6 months after a paclitaxel-eluting stent to allow coincident administration of aspirin and clopidogrel. If surgery is anticipated in the near future, simple balloon angioplasty may be preferable. Where patients have been successfully revascularized and convalesced, if they remain asymptomatic, risk for perioperative myocardial infarction should be reduced for 5 years.

In general, coronary artery bypass grafting would not be employed for a right coronary artery lesion, such as the one in this patient.

#### **REFERENCES (20)**

- Adesanya AO, de Lemos JA, Greilich NB, et al. Management of perioperative myocardial infarction in noncardiac surgical patients. *Chest* 2006; 130:584-596.
- Ashton MC, Petersen NJ, Wray NP, et al. The incidence of perioperative myocardial infarction in men undergoing noncardiac surgery. *Ann Intern Med* 1993; 118:504-510.
- Eagle KA, Berger PB, Calkins H, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery-executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* 2002; 39:542-553.
- Eagle KA, Rihal CS, Mickel MC, et al. Cardiac risk of noncardiac surgery: Influence of coronary disease and type of surgery in 3368 operations. *Circulation* 1997; 96:1882-1887.
- Landesberg G, Shatz V, Akopnik I, et al. Association of cardiac troponin, CK-MB, and postoperative myocardial ischemia with long-term survival after major vascular surgery. *J Am Coll Cardiol* 2003; 42:1547-1554.

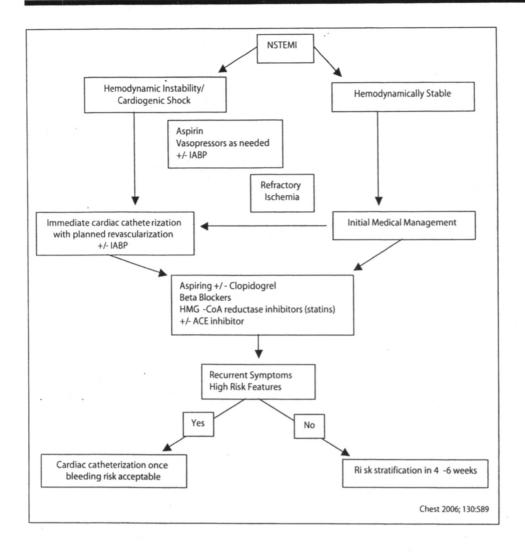
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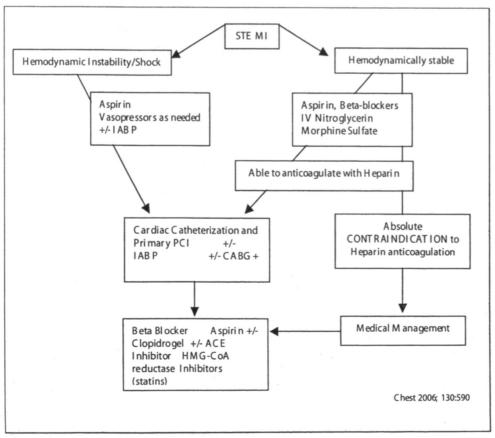
Answer: C

Older studies report that perioperative myocardial infarction is typically seen in the first 3 postoperative days, when patients mobilize fluids administered in the operating room and thrombotic risk is increased. More recent data suggest that perioperative myocardial infarction may begin with events occurring on the day of surgery or the day following this procedure. Typically, myocardial infarction following surgery is silent and of the non-ST-segment elevation type. Perioperative pain is an important contributing factor, as it is frequently associated with hypertension, tachycardia, and catecholamine surge. All of these factors can lead to supply demand mismatch in the myocardium supply demand mismatch, with respect to oxygen transport in the myocardium. In pathologic studies, patients with tight, mature coronary stenoses are less likely to thrombose a coronary artery than patients with less mature coronary lesions. While rupture of less mature coronary plaques is thought important in perioperative myocardial infarction, ischemia secondary to inadequate myocardial blood supply is also thought to be an important contributing factor.

While this patient is young, and there is no history of preexisting coronary disease, a long difficult operation with blood loss warranting significant transfusion places the patient at increased risk. In the absence of ST-segment elevation and stable hemodynamics, appropriate supportive medical therapy begins with aggressive pain relief to blunt hypertension, tachycardia, and sympathetic stimulation. β-Blockade is appropriate in the absence of hypotension, and bradycardia as manifest by HR in the range of 50/min. Patients with extensive dissection and increased bleeding risk are not good candidates for low-molecular-weight heparin. If heparin is employed in this patient, unfractionated heparin should be used due to ease of reversibility with protamine. Of the various antiplatelet agents available, aspirin is the safest option to employ, because it inhibits only one of the pathways (thromboxane A2), leading to platelet aggregation. Other platelet inhibitors, including glycoprotein IIb, IIIa platelet receptor antagonists, are more effective platelet aggregation inhibitors and should be avoided in the perioperative setting due to an increased risk of major bleeding. ACE-inhibitors should be employed if not contraindicated by known low left ventricular ejection fraction or hypotension associated with their use.

If ST-segment elevation is noted on electrocardiogram, coronary vasodilatation with nitroglycerin and intraaortic balloon counterpulsation may be beneficial in improvement of myocardial oxygen delivery. Due to associated bleeding risk, fibrinolytic therapy is not indicated, even in these patients who are sometimes desperately ill. Percutaneous coronary interventions with balloon angioplasty, preferably without stent deployment, is recommended if the patient may receive at least some antiplatelet therapy. Finally, use of a statin (HMG CoA-reductase inhibitor) may be considered, as these agents reduce the incidence of recurrent ischemic events and cardiac death. See figures below.





#### **REFERENCES (21)**

- Adesanya AO, de Lemos JA, Greilich NB, et al. Management of perioperative myocardial infarction in noncardiac surgical patients. *Chest* 2006; 130:584-596.
- Bertrand ME, Simoons ML, Fox KA, et al. Management of acute coronary syndromes: Acute coronary syndromes without persistent ST segment elevation: recommendations of the Task Force of the European Society of Cardiology. *Eur Heart J* 2000; 21:1406-1432.
- CAPRIE Steering Committee. A randomised, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). *Lancet* 1996; 348:1329-1339.
- Lau J, Antman EM, Jimenez-Silva J, et al. Cumulative meta-analysis of therapeutic trials for myocardial infarction. N Engl J Med 1992; 327:248-254.

RATIONALE (22)

Answer: E

This patient presents with tracheal laceration associated with intubation for her laparoscopic procedure. Typically, these lesions involve the posterior membranous portion of the trachea and are situated above the carina. Surgical repair is the historical standard, with direct suture repair of the trachea and drainage of the mediastinum, as necessary. A large recent series, however, suggests that less invasive strategies may offer equivalent results with lesser morbidity. In a recent series of 30 patients, the majority of individuals treated for this problem in a regional referral center were managed with stenting using the endotracheal tube, permitting continued spontaneous breathing or low levels of noninvasive ventilation. Mortality in this series, which included many desperately ill patients, was 20%, but only half of the patients involved who died did so with pulmonary complications. In cases where bronchoscopy revealed lesions close to the carina, a double lumen endotracheal tube was required. Half of the involved patients did not require mechanical ventilation and were managed without thoracotomy.

Tube thoracostomy is not indicated, unless an obvious pneumothorax is present.

# **REFERENCES (22)**

- Conti M, Pougeoise M, Wurtz A, et al. Management of postintubation tracheobronchial ruptures. Chest 2006; 130:412-418.
- Marty-Ane CH, Picard E, Jonquet O, et al. Membranous tracheal rupture after endotracheal intubation. *Ann Thorac Surg* 1995; 60:1367-1371.
- Massard G, Rouge C, Dabbagh A, et al. Tracheobronchial lacerations after intubation and tracheostomy. *Ann Thorac Surg* 1996; 61:1483-1487.
- Marquette CH, Bocquillon N, Roumilhac D, et al. Conservative treatment of tracheal rupture. *J Thorac Cardiovasc Surg* 1999; 117:399-401.
- Ross HM, Grant FJ, Wilson RS, et al. Nonoperative management of tracheal laceration during endotracheal intubation. *Ann Thorac Surg* 1997; 63:240-242.

RATIONALE (23)

Answer: C

Patients with acute abdominal pain present with sudden or gradual onset of discomfort. Sudden abdominal pain may develop as a consequence of rupture of a hollow viscus, aortic or visceral artery aneurysm rupture, or critical expansion of a structure in the abdomen. Gradual or nonspecific abdominal pain may begin with an inflammatory process, such as appendicitis or diverticulitis.

Peritonitis is associated with irritation of the peritoneal lining. The peritoneum has visceral and parietal surfaces. Pain is generally better localized when a portion of the parietal, rather than visceral, peritoneum is involved in the intraabdominal process. Thus, the classic presentation of appendicitis is periumbilical abdominal pain (visceral localization of the midgut), which progresses to right lower quadrant discomfort (parietal innervation which is more localized). Generalized peritonitis is cause by irritation of the entire abdominal lining and is more likely to result in a surgical emergency.

If findings of diffuse peritonitis are elicited, little diagnostic testing is necessary. Routine laboratory tests are sent with an upright chest radiograph to enhance preoperative preparation. Upright chest radiograph is a rapid screen to determine if there is significant free air in the abdomen. A CT scan is often obtained prior to or with surgical consultation. With a clinical diagnosis of generalized peritonitis, however, operative therapy should not be delayed for additional imaging without the consent of the surgeon. Patients with significant comorbidities (and hemodynamic stability) may benefit from this imaging, if operative planning is optimized. A nondiagnostic CT scan, however, should not deter from operation.

#### **REFERENCE (23)**

Jones RS, Claridge JA. Acute abdomen. In: Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery: The Biological Basis of Modern Surgical Practice. Philadelphia, PA: Elsevier Saunders; 2004:1219-1239.

RATIONALE (24)

Answer: C

Pressure-induced rhabdomyolysis is a rare but well described postoperative complication resulting from prolonged unrelieved pressure to muscle during surgery. In the morbidly obese patient, major risk factors include prolonged operative time and patient weight. Rhabdomyolysis after bariatric surgery may affect lower limbs, gluteal, or lumbar regions.

The most common presentation of rhabdomyolysis in the setting of bariatric surgery is numbness and muscular pain. Cutaneous changes may occur over sites of muscle injury.

Treatment consists of aggressive hydration, mannitol administration to mobilize muscular interstitial fluid, and increased renal tubular flow and alkalinization of urine with sodium bicarbonate to increase the solubility of myoglobin. Fasciotomy is not routinely employed.

Acute renal failure (prevalence 50%) results from hypovolemia, tubular obstruction, acidosis, and free-radical release. Recovery of tubular function is common though a period of renal replacement therapy may be required.

#### **REFERENCES (24)**

Khurana RN, Baudendistel TE, Morgan EF, et al. Postoperative rhabdomyolysis following laparoscopic gastric bypass in the morbidly obese. *Arch Surg* 2004; 139:73-76. Pieracci FM, Barie PS, Pomp A. Critical care of the bariatric patient. *Crit Care Med* 2006; 34:1796-1804.

RATIONALE (25)

Answer: A

Perioperative risk depends on comorbid conditions, such as ischemic heart disease, obstructive pulmonary disease, and diabetes. In addition, surgery that is emergent, prolonged, and with more blood loss is associated with poorer outcome. Exercise tolerance provides an estimate of cardiorespiratory reserve. An ASA score could also be estimated from the information and used to describe the physical status of the patient. See table below.

ASA Score	Physical Status	
1	A normal healthy patient	
2	A patient with a mild systemic disease	
3	A patient with a severe systemic disease that limits activity, but is not incapacitating	
4	A patient with an incapacitating systemic disease that is a constant threat to life	
5	A moribund patient not expected to survive 24 hours with or without operation	

Patient A would be classified as ASA V, because without surgery, the patient would not survive. The type of surgery would also be prolonged and associated with large volumes of blood loss. These factors outweigh the relatively mild comorbidity, good exercise tolerance, and younger age. Patient D would have the next highest risk due to the emergent surgery and moderate exercise tolerance, but the surgery would not be anticipated to be prolonged or result in significant blood loss. Patient B has the lowest risk due to the local surgery. Patient C has a slightly higher risk than Patient B due to the longer intracavitary procedure and general anesthesia.

#### **REFERENCES (25)**

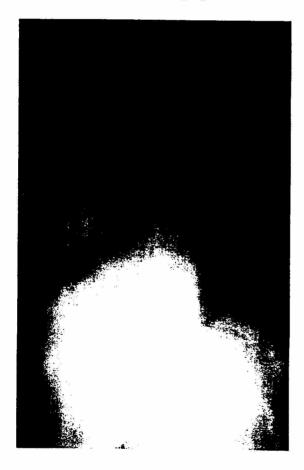
- ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery. January 2002. Available at: http://www.americanheart.org/presenter.jhtml?identifier=3036136. Accessed June 12, 2007.
- Lee TH, Marcantonio ER, Mangione CM, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation* 1999; 100:1043-1049.
- Older P, Smith R, Courtney P, et al. Preoperative evaluation of cardiac failure and ischemia in elderly patients by cardiopulmonary exercise testing. *Chest* 1993; 104:701-704.

# SECTION 15: Trauma/Environmental Injury

# SECTION 15: TRAUMA/ENVIRONMENTAL INJURY

Instructions: For each question, select the most correct answer.

1. A 46-year-old man is admitted to the ICU after a motor vehicle crash. He has a flail chest and pelvic fractures. He receives ventilation through an emergent tracheostomy due to facial injuries. The patient has a long history of smoking. He is not bleeding and has been hemodynamically stable. Over the next 24 hours, he develops increasing pulmonary artery pressures. His chest radiograph is shown below.



Which one of the following is the most likely cause of his acute pulmonary artery hypertension?

- A. Right heart contusion
- B. Pulmonary contusion
- C. Fat embolism
- D. Thromboembolism
- E. Exacerbation of chronic obstructive pulmonary disorder

2.	In patients with blunt trauma, what is the most common chest injury?				
	A. Rib fractures				
	B. Pneumothorax				
	C. Hemothorax				
	D. Pulmonary contusion				
-					
3.	A 24-year-old female restrained driver is injured in a motor vehicle crash. She is admitted to the ICU after a negative laporatomy, rodding of her femur fracture, and placement of an external fixator on her pelvic fracture. She is intubated and ventilated with good breath sounds bilaterally. The chest radiograph is otherwise unremarkable. Packed red blood cells and crystalloid infusion initially normalized her blood pressure, but hypotension occurs.				
	Which one of the following is most appropriate at this time?				
	A. Pelvic arteriogram				
	B. Return to operating room				
	C. Placement of pulmonary artery catheter				
	D. Transesophageal echocardiogram				
4.	Which of the following statements is most correct regarding antibiotic prophylaxis in penetrating bdominal trauma?				
	A. Allows avoidance of abscess formation with fecal spillage B. Reduces the incidence of wound infection				
	C. Antibiotic prophylaxis is not routinely employed in trauma				

D. Requires multiple drugs if bowel perforation is detected

E. Must include coverage of Gram-negative aerobes and cutaneous organisms

5. A young male sustains a gunshot wound and presents hypotensive with multiple small bowel and colonic injuries. Primary suture repair is performed. Blood loss is described as significant, and 4 U of packed red blood cells are given. The patient is hemodynamically stable on arrival in the ICU.

Which of the following is the appropriate duration of perioperative antibiotic coverage?

- A. 1 dose at incision
- B. 24 hours
- C. 48 hours
- D. 7 days
- E. 10 days
- 6. IV morphine 4 mg every 3 hours fails to relieve the leg pain of a 20-year-old male who had a gunshot wound to the leg with femoral artery and vein injury. He had arterial repair and venous ligation. In spite of elevation of his leg, there is swelling from the thigh to the foot. Passive movement of his foot causes profound calf pain. He has palpable pulses at the posterior tibal and dorsalis pedis, and his ankle brachial index (ankle blood pressure divided by the brachial blood pressure) is normal.

Which one of the following should you do?

- A. Increase the amount of pain medicine
- B. Obtain an arteriogram
- C. Perform fasciotomy
- D. Place an epidural catheter for pain control
- E. Anticoagulate the patient
- 7. Which of the following statements regarding blunt aortic injury is most correct?
  - A. Occurs more frequently in head-on versus side impact high-speed motor vehicle crashes
  - B. May be excluded by a normal chest radiograph
  - C. The majority of patients sustaining this injury die in hospital
  - D. Requires early-operative repair once the diagnosis is made

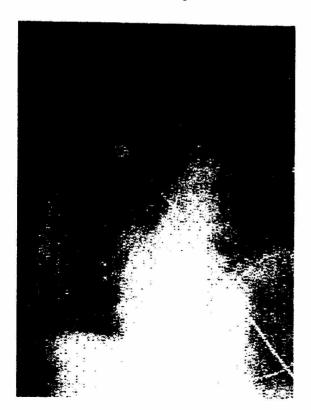
8. A chest tube is placed for hemothorax and 1,000 mL of blood is evacuated after a stab wound.

Which of the following is the most correct antibiotic strategy to decrease the incidence of empyema?

- A. Gram-positive coverage for the duration of placement of chest tube
- B. Gram-positive coverage with 1 dose prior to chest tube insertion
- C. Antibiotics are not indicated
- D. Broad-spectrum coverage for Gram-positive and Gram-negative organisms for 24 hours
- E. Gram-positive coverage for 24 hours beginning at time of tube insertion
- 9. A 19-year-old female is admitted to the ICU with a history of having been kicked in the upper abdomen by a horse. This radiograph below was obtained.

Which one of the following statements is most correct?

- A. This condition is rarely associated with cardiopulmonary compromise
- B. This condition requires immediate surgery
- C. The diagnosis is usually made with chest radiograph
- D. A chest tube should be placed



- 10. Which one of the following best predicts effective resuscitation from injury associated with blood loss?
  - A. Improving mental status
  - B. Blood pressure 130/90 mm Hg
  - C. Normalizing lactate level
  - D. Urine output 80 mL/h
- 11. Which one of the following is not associated with increasing base deficit and risk for morality in trauma patients?
  - A. Occurrence of ARDS
  - B. Increasing mortality
  - C. Decreasing mixed venous oxygen saturation
  - D. Increasing IL-6 level
  - E. Increasing neutrophil CD11B expression
- 12. A 65-year-old male presents after a high-speed motor vehicle crash with a widened mediastinum on chest radiograph, and aortic injury is diagnosed. He is noted to have an open femur fracture, peritoneal signs, and hypotension. Glasgow Coma Scale Core deteriorates to 6, and the patient is intubated.

Which one of the following treatments is of highest priority?

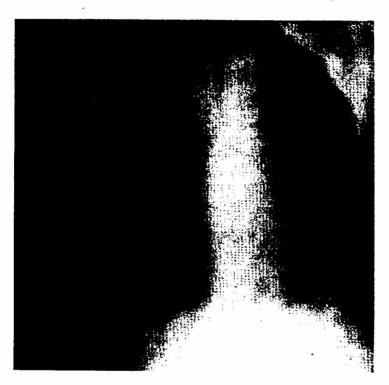
- A. Intracranial pressure control for traumatic brain injury prior to operation
- B. Laparotomy for abdominal catastrophe
- C. Repair of aortic injury
- D. Continued resuscitation as the patient cannot tolerate anesthesia safely
- E. Immediate repair of open femur fracture

13. An 18-year-old male is admitted to the ICU after an operation to repair injuries from a gunshot wound. He had injuries to the vena cava, aorta, duodenum, liver, colon, and small bowel. During his operation, he received 30 U of packed cells, 5 U of fresh frozen plasma, and 10 U of platelets. On admission to the ICU, his pulse is 120/min, blood pressure is 100/68 mm Hg, and temperature is 33°C (91.4°F). He is intubated and receives ventilation with O<sub>2</sub> sat of 96%. He has bleeding from his midline incision and IV sites.

Which one of the following is most appropriate to control bleeding?

- A. Reoperation
- B. Desmopressin acetate
- C. Angiographic embolization
- D. Heparin
- E. Rewarming
- 14. Which one of the following is most predictive of the need for angiography in the early management of pelvic fractures?
  - A. Anteriorly displaced fracture through the pubic symphysis
  - B. Vertical shear through the hemipelvis
  - C. Gross blood on peritoneal cavity aspiration
  - D. Massive posterior fractures including the sacrum
  - E. Contrast extravasation in the retroperitoneum on CT scan of the pelvis
- 15. Which one of the following is *least predictive* of the need for laparotomy in patients with pelvic fractures associated with massive retroperitoneal hematoma?
  - A. Involuntary guarding
  - B. Large perihepatic fluid collection on abdominal ultrasound
  - C. Large retroperitoneal fluid collection on CT scan
  - D. Hypotension after successful embolization of the pelvis and placement of an external fixator

- 16. A young man presents after a high speed automobile crash. Which one of the following statements is most correct concerning the chest radiograph shown below?
  - A. Laparotomy for obvious abdominal hemorrhage should be delayed pending further diagnostic chest studies
  - B. The most common cause of this chest radiographic abnormality is radiographic technique
  - C. A normal ECG excludes significant cardiovascular injury
  - D. Tracheostomy should be performed as soon as possible



- 17. Which one of the following is most correct concerning inhalation injury?
  - A. Inhalation injury exhibits characteristic radiograph changes
  - B. Early mortality in inhalation injury is due to airway obstruction
  - C. Inhalation injury typically occurs when burn injury occurs in a closed space
  - D. Inhalation injury typically occurs with heat exposure to the lungs and large airways

- 18. Which one of the following statements concerning hyperbaric oxygen in burn and inhalation injury is most correct?
  - A. Is valuable in patients with isolated carbon monoxide toxicity and mental status changes
  - B. Should be considered to support care in every large burn particularly with inhalation injury
  - C. Has no role in the management of smoke inhalation or large burns
  - D. Is useful in burn wound infection due to Pseudomonas
- 19. A young male sustains a 50% total body surface area of 2nd- and 3rd-degree burns with involvement of the torso and legs. Inhalation injury is present.

Which one of the following statements is most correct concerning resuscitation?

- A. A pulmonary artery catheter will optimize fluid administration
- B. Restriction of resuscitation fluids is needed to minimize pulmonary edema
- C. Supplemental colloids are essential in the first 24 hours
- D. This combination of injuries may require fluid administration in excess of normal resuscitation formula predictions
- 20. Which one of the following statements is most correct regarding initial management of brain injury?
  - A. All patients with moderate brain injury require intubation (Glasgow Coma Score <12)
  - B. Extracranial injuries play a role in determining outcome of traumatic brain injury, as they may affect optimal oxygenation or perfusion
  - C. Fluid administration is minimized, as long as systolic blood pressure is >90 mm Hg and Pao<sub>2</sub> >60 mm Hg
  - D. Hypotension does not exclude the use of mannitol

21. Three days after severe brain injury, a young male is giv	en a neuromuscular blocker to facilitate
management of ventriculostomy-monitored intracranial	pressure.

Which one of the following offers the best nutrition support?

- A. Replacement of 80% of resting metabolism expenditure
- B. Replacement of 100% of resting metabolism expenditure
- C. Replacement of 120% of resting metabolism expenditure
- D. Replacement of 140% resting metabolism expenditure
- 22. A 28-year-old male suffers severe blunt head trauma. On arrival to the emergency department, the airway is patent and breathing and circulation are intact. The patient opens eyes only to painful stimuli. He moans unintelligible sounds. Decorticate posturing is observed. Pupils are midrange, symmetric, and sluggish.

Which one of the following statements is most correct?

- A. The patient should be immediately transported to the radiology department for a CT scan of the head
- B. Endotracheal intubation should be avoided at this point because the procedure will elevate intracranial pressure
- C. Immediate endotracheal intubation should be performed in the emergency department
- D. The patient should be endotracheally intubated with inline stabilization and hyperventilated to a Pco<sub>2</sub> target of 28 mm Hg
- 23. Which one of the following statements regarding management of cerebral perfusion pressure is least correct?
  - A. Cerebral perfusion pressure is optimally ≥70 mm Hg
  - B. Mortality increases by as much as 20% with reduction of cerebral perfusion pressure, from 70 mm Hg to 60 mm Hg
  - C. Raising mean arterial pressure is expected to increase intracranial pressure
  - D. It may be necessary to increase mean arterial pressure to >100 mm Hg

24. A 63-year-old male suffers severe blunt head trauma in a high-speed motor vehicle collision. Upon arrival to the emergency department, the airway is patent, and breathing and circulation are intact. The patient's eyes open spontaneously. He is talking but confused. He does not follow commands but demonstrates intentional avoidance of painful stimuli. No other injuries are found in his emergency department evaluation. A noncontrast CT scan is performed and interpreted as negative. He is transported to the ICU for further monitoring.

Six hours after his arrival to the ICU, the patient's neurologic status begins to deteriorate. The patient's eyes are closed, but he opens his eyes to painful stimuli. He has no verbal response. There is spontaneous movement of the extremities, but he does not follow commands. Pupils are midrange, symmetric, and sluggish. The patient is intubated and mechanical ventilation is initiated.

Which one of the following statements is most correct?

- A. An intraventricular catheter (or other technique of intracranial pressure monitoring) is only indicated if repeat, noncontrast, CT scan shows evidence of increased intracranial pressure
- B. A CT scan with IV contrast is indicated for this patient to evaluate for diffuse axonal injury
- C. The patient should not receive anticonvulsants at this time
- D. Administration of steroids (dexamethasone) in this patient will improve neurologic outcome
- E. Radiographic manifestation of intraparenchymal brain hemorrhage may be delayed up to 24 hours after the primary brain injury

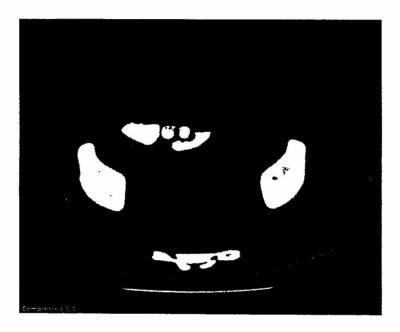
25. A farmer presents with this wound (below) minutes after being caught in a combine. The patient is alert with a blood pressure of 120/80 mm Hg and a pulse rate of 95/min. Mental status is good.



Which one of the following statements best describes the state of this patient?

- A. This wound is contaminated, but the patient is not yet infected
- B. Immediate administration of antibiotics is essential
- C. In a fully immunized patient, tetanus immune globulin is required
- D. Advanced age and a history of lymphoma would change antimicrobial therapy

26. A 15-year-old male presents 3 weeks after a high-grade splenic injury associated with significant pelvic hematoma formation. The splenic injury was embolized, and the patient did not require surgery. At the time of his return for evaluation, the patient had lost 15 lb and had intermittent fevers. He reported difficulty moving his bowels, and one bowel movement was described as grossly bloody. CT scan obtained 3 weeks after injury is shown below. There are no cutaneous signs of injury.



Which of the following choices represents optimum management for this patient?

- A. This patient has recurrent pelvic hemorrhage, which requires percutaneous drainage or operative exploration
- B. This patient has a pelvic abscess, which mandates immediate operation intervention
- C. A diagnostic percutaneous aspirate should be performed to rule out infection and, if positive results are found, followed by definitive drainage
- D. Immediate percutaneous drainage of the entire fluid collection, if possible, is warranted

27. A 24—year-old male presents to the ICU with severe traumatic brain injury (Glasgow Coma Scale Score = 6) and extremity injuries including a severe pelvic fracture that is hypotensive. The surgeons are preparing to take the patient to the operating room to place an external fixator and stabilize the pelvis.

In addition to optimizing mean arterial pressure, which one of the following is most appropriate?

- A. Place an intracranial pressure monitor and transfuse to a hemoglobin ≥10 mg/dL
- B. Transfuse to a hemoglobine ≥10 mg/dL. Intracranial pressure monitor is not needed
- C. Transfuse to a hemoglobin >7 10 mg/dL and place an intracranial pressure monitor.
- D. Transfuse to a hemoglobin ≥7 mg/dL. Intracranial pressure monitoring is not needed
- 28. A 16-year-old female suffers 60% total body surface area burns and inhalation injury in a car fire. Emergency medical services intubate the patient, and she is transferred to your burn center. She is hypoxemic with bilateral pulmonary infiltrates. You are the critical care consultant.

What are your recommendations for optimal respiratory support?

- A. Early (<48 hours) tracheostomy and high frequency oscillatory ventilation
- B. High-frequency oscillatory ventilation and tracheostomy, if needed later in the hospital course
- C. Low tidal volume ventilation and early (<48 hours) tracheostomy
- D. Low tidal volume ventilation and tracheostomy, if needed later in hospital course
- 29. An 80-year-old female suffers femur fracture in a motor vehicle crash. She undergoes intramedullary rodding and is initially extubated in the postanesthesia care unit. Within an hour, she is reintubated with hypoxemia and progressive pulmonary infiltrates and mental status changes.

On arrival in the ICU, your examination confirms the above and identifies a petechial rash at the torso and neck. The Glasgow Coma Scale Score is 4.

Which of the following is the most appropriate intervention?

- A. Low tidal volume ventilation with positive end expiratory pressure
- B. Heparinization after CT scan of the head is negative for stroke or intracranial hemorrhage
- C. High-dose steroids
- D. Bronchoscopy for evacuation of lipid-laden macrophages

30. A 21-year-old patient is brought to the emergency department after a gunshot wound to the proximal thigh. He has an estimated 1,000-mL blood loss. In the trauma bay, the patient's HR is 120/min; blood pressure is 117/95 mm Hg; RR is 25/min. He appears to be mildly anxious. He appears to have an arterial source of bleeding from a direct injury to the femoral artery. Direct pressure is applied which appears to markedly slow the blood loss.

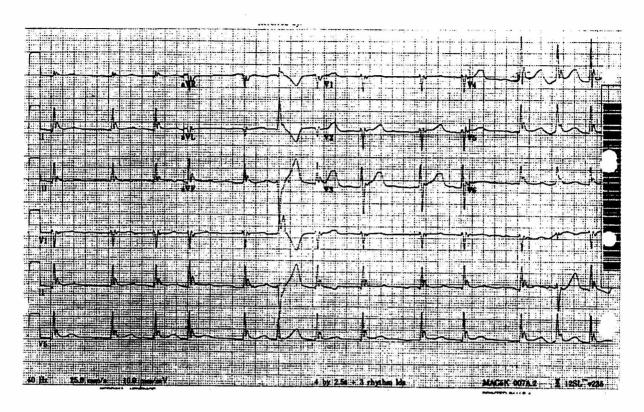
Which one of the following statements is most correct?

- A. Type-specific blood should be administered immediately
- B. The absence of hypotension excludes the possibility of shock
- C. The lack of a widened pulse pressure excludes shock
- D. The patients resuscitation fluid should be limited to crystalloid at this time
- 31. A 31-year-old male is brought to a trauma center after suffering severe blunt chest and abdominal trauma in a high-speed motor vehicle collision. On arrival to the trauma bay, the patient is already intubated and bag ventilated. His BP is 80/40 mm Hg, HR is 125/min. The breath sounds are decreased on the left side of the chest with crepitations on palpation of the chest wall. A chest tube is promptly inserted in the left chest with evacuation of air and a small amount of blood. The BP rises to 100/60 mm Hg. Two large bore IVs have been inserted into both arms, and crystalloid resuscitation is ongoing. A chest radiograph shows an expanded lung and a well-placed chest tube as well as bilateral rib fractures and extensive bilateral pulmonary contusions. A pelvis radiograph does not show evidence of fracture. A secondary survey is significant for heart sounds that are difficult to auscultate and a firm slightly distended abdomen. The patient's systolic BP drops down again into the mid-80 mm Hg-range. Crystalloid resuscitation is ongoing, and type-specific uncross-matched blood will be administered.

Which of the following should be performed next?

- A. Computerized tomography scan of the abdomen
- B. Exploratory laparotomy
- C. Focused assessment sonography in trauma
- D. Repeat chest radiograph
- E. Aortogram

32. A patient is brought to the emergency department with altered mental status. An ECG is obtained (shown below). Which of the following interventions should be performed to correct the ECG abnormality?



- A. Rewarming
- B. Thrombolytics
- C. Amiodarone
- D. Activated charcoal
- E. Sodium bicarbonate

# SECTION 15: TRAUMA/ENVIRONMENTAL INJURY

#### **ANSWERS:**

1-B; 2-A; 3-A; 4-B; 5-B; 6-C; 7-D; 8-E; 9-C; 10-C; 11-C; 12-B; 13-E; 14-E; 15-C; 16-B; 17-C; 18-A; 19-D; 20-B; 21-D; 22-C; 23-C; 24-E; 25-A; 26-D; 27-C; 28-D; 29-A; 30-D; 31-C; 32-A

RATIONALE (1) Answer: B

In this injury scenario, either pulmonary contusion or right heart contusion should be considered as a potential cause of acute right heart failure. The chest radiograph is most compatible with pulmonary contusion. The initial ECG reveals sinus tachycardia only, and the delayed manifestation of right heart failure go against right heart contusion. Right heart failure is caused by an acute increase in right ventricular afterload or sudden increase in pulmonary artery pressures. Because the right ventricle has a thin muscular wall, it cannot generate the force necessary to overcome the pulmonary pressures, and volume accumulates in the right ventricle. This dilates the right side of the heart and encroaches on the left heart. Causes of right heart failure include acute respiratory distress syndrome, pulmonary embolus, pulmonary contusion, chronic obstructive pulmonary disorder, positive end expiratory pressure, increased mean airway pressures, ischemia, and direct right heart contusion. Chronic obstructive pulmonary disorder-induced cor pulmonale would be present at the time of presentation. Although fat emboli and pulmonary thrombolism may produce acute pulmonary artery hypertension, the timing of this presentation makes these diagnoses less likely, and the chest radiograph is most compatible with lung contusion. Also, pelvic fractures, in the absence of long bone fractures, would be unlikely to produce fat emboli.

#### REFERENCES (1)

Clark GC, Schecter WP, Trunkey DD. Variables affecting outcome in blunt chest trauma: Flail chest vs pulmonary contusion. *J Trauma* 1988; 28:298-304.

Hoff ST, Shots SD, Eddy VA, et al: Outcome of isolated pulmonary contusion in blunt trauma patients. *Am Surg* 1994; 60:138-142.

Pretre R, Chilcott M. Blunt trauma to the heart and great vessels. N Engl J Med 1997; 336:626-632. Velmahos GC, Karaiskakis M, Salim A, et al. Normal electrocardiography and serum troponin I levels preclude the presence of clinically significant blunt cardiac injury. J Trauma 2003; 54:45-51.

RATIONALE (2)

Answer: A

While pulmonary contusion is a common chest injury from blunt trauma, rib fractures are more common (although not necessary for contusion to occur). There is usually a pulmonary contusion associated with rib fractures. Rib fractures occur in about 48% of patients with chest trauma. Generally, pneumothorax and hemothorax are complications of rib fractures. Flail chest is associated with multiple segmental rib fractures causing an unstable chest wall. There is usually an associated pulmonary contusion. The hallmark of pulmonary contusion is progressive hypoxemia. The initial chest radiograph is normal in one third of patients with pulmonary contusion and will worsen by 6 to 24 hours. Unless an arterial blood gas measurement is obtained, the hypoxemia may not be recognized. The Pao<sub>2</sub> will worsen over the first 24 to 48 hours, as the edema around the contusion increases. A CT scan of the chest will show the extent of the contusion. Intubation and ventilation are required for patients with progressive hypoxemia, and initial radiographic evidence of pulmonary contusion.

#### **REFERENCES (2)**

Allen GS, Coates NE. Pulmonary contusion: a collective review. Am Surg 1996; 62:895-900. Cohn SM. Pulmonary contusion: review of the clinical entity. J Trauma 1997; 42:973-979. Kulshrestha P, Munshi I, Wait R. Profile of chest trauma in a level I trauma center. J Trauma 2004; 57:576-581.

Miller PR, Croce MA, Bee TK, et al. ARDS after pulmonary contusion: accurate measurement of contusion volume identifies high-risk patients. *J Trauma* 2001; 51:223-230.

RATIONALE (3)

Answer: A

The majority of patients with major pelvic fractures require blood transfusion. The amount of bleeding varies with the type of pelvic fracture. Patients with disruption of both anterior and posterior elements of the pelvis have an unstable pelvis and have profuse bleeding. The majority of the bleeding occurs from the pelvic venous plexus in the retroperitoneum. A large potential space from the diaphragm to the thigh opens. This space can accommodate many units of blood. Stabilization and/or compression of the pelvis will control this type of bleeding. The 5 to 10% of patients who remain hemodynamically unstable after pelvic stabilization are usually bleeding from major branches of the internal iliac arteries. This bleeding is best controlled by angiographic embolization. Surgical ligation is usually unsuccessful. Packing at the time of surgery with stabilization of the pelvis is often followed by angiographic embolization. Pulmonary artery catheters and the transesophageal echocardiogram can be used if the diagnosis of the type of shock is in doubt. Bilateral chest tubes are not indicated in the absence of bilateral hemo/pneumo thoracices.

#### **REFERENCES (3)**

- Blackmore CC, Cummings P, Jurkovich GJ, et al. Predicting major hemorrhage in patients with pelvic fracture. *J Trauma* 2006;61:346-352.
- Blackmore CC, Jurkovich GJ, Linnau KF, et al. Assessment of volume of hemorrhage and outcome from pelvic fracture. *Arch Surg* 2003; 138:504-509.
- Brasel KJ, Pham K, Yang H, et al. Significance of contrast extravasation in patients with pelvic fracture. *J Trauma* 2007;62:1149-1152.
- Cryer HM, Miller FB, Evers BM, et al. Pelvic fracture classification: correlation with hemorrhage. *J Trauma* 1988; 28:973-980.
- Flint L, Babikian G, Anders M, et al. Definitive control of mortality from severe pelvic fracture. *Ann Surg* 1990; 211:703-707.
- Kimbrell BJ, Velmahos GC, Chan LS, et al. Angiographic embolization for pelvic fractures in older patients. *Arch Surg* 2004; 139:728-733.
- Moreno C, Moore EE, Rosenberger A, et al. Hemorrhage associated with pelvic fracture: a multispeciality challenge. *J Trauma* 1986; 26:987-994.

RATIONALE (4)

Answer: B

The importance of broad-spectrum antibiotic coverage for patients with penetrating abdominal trauma was demonstrated in the 1970s. Antibiotic regimens, which included coverage of aerobes and anaerobes, have the smallest number of postoperative infections. Prophylactic antibiotics for patients sustaining penetrating abdominal injuries with intestinal contamination have a role for reducing the rate of *incisional wound infection* due to gastrointestinal soiling. A single dose providing sufficient concentration within the wound during the vulnerable period is optimal. There are sufficient data to recommend a single preoperative dose of prophylactic antibiotics with broad-spectrum aerobic and anaerobic coverage as a standard-of-care for trauma patients sustaining penetrating abdominal wounds. In the absence of hollow viscus injury, no further administration of antibiotics is required.

### **REFERENCES (4)**

- Dellinger EP. Antibiotic prophylaxis in trauma: penetrating abdominal injuries and open fractures. Reviews of Infectious Diseases 1991; 13:S847-S857.
- Hofstetter SR, Pachter HL, Bailey AA, et al. A prospective comparison of two regimens of prophylactic antibiotics in abdominal trauma: cefoxitin versus triple drug. *J Trauma* 1984; 24:307-310.
- The EAST Practice Management Guidelines Work Group. Practice management guidelines for prophylactic antibiotic use in penetrating abdominal trauma. Available at: www.east.org/tpg.html. Accessed June 11, 2007.

RATIONALE (5)

Answer: B

Antibiotic regimens, which include coverage of aerobes and anaerobes, have the smallest number of postoperative infections. The other aspect of prophylactic antibiotic administration in trauma is a potential therapeutic role. The problem is to define the time period when contamination of the abdominal cavity becomes an established infection. At celiotomy, the intestinal wound is closed eliminating further contamination and soiling of the peritoneal cavity. Therefore, further antibiotic administration should not be necessary. A recent metaanalysis reviewed 17 studies assessing the effectiveness of single agent versus combination therapy containing aminoglycosides for penetrating wounds. This report concluded that single  $\beta$ -lactam agents were as effective as combination therapy. Prophylactic antibiotics are required for only 24 hours in the presence of injury to any hollow viscus.

#### **REFERENCES (5)**

Fabian TC, Croce MA, Payne LW, et al. Duration of antibiotic therapy for penetrating abdominal trauma: A prospective trial. Surgery 1992; 112:788-795.

Fabian TC, Hoefling SJ, Strom PR, et al. Use of antibiotic prophylaxis in penetrating abdominal trauma. Clin Ther 1982; 5:38-47.

The EAST Practice Management Guidelines Work Group. Practice management guidelines for prophylactic antibiotic use in penetrating abdominal trauma. Available at: www.east.org/tpg.html. Accessed June 11, 2007.

RATIONALE (6)

Answer: C

This patient has compartment syndrome that occurs in the extremity from increased pressure in a closed fascial muscle compartment. The increased pressure causes small outflow veins to collapse associated with increased swelling, arterial thrombosis, and nerve and muscle death. Venous hypertension from femoral vein ligation has caused this patient's compartment syndrome.

Elevation of the involved extremity eliminates the need for fasciotomy in 80% of patients but has failed in this case. Bleeding into the compartment, crush injury, infection, edema, snakebites, and ischemia reperfusion injury are other causes of compartment syndrome. In the awake patient, pain is the most frequent symptom (ie, pain out of proportion to the injury). Pain serves as warning sign that the leg needs to be examined for other signs of compartment syndrome. Both active and passive muscle activity increase pain. Paraesthsias (web space between the first and second toe in lower extremity) are often rapidly followed by paralysis. The presence or absence of the pulse does not correlate with compartment syndrome.

Compartment pressures can be measured by manometery or commercial devices. Compartment pressures >30 mm Hg (exceeding capillary perfusion pressure) must be addressed. Elevation of the extremity is the first treatment, followed quickly by fasciotomy. Early fasciotomy to decompress the compartment will prevent neuromuscular deficits.

Adequate pain control is important but does not take the place of recognizing compartment syndrome. The ankle-brachial index (ABI) is obtained by measuring the blood pressure in the posterior tibal artery and the brachial artery and is calculated using ankle blood pressure divided by the brachial blood pressure. A normal ABI is 0.9 to 1.0. The further the ABI is from 1.0, the more arterial disease is present. Because a pulse with normal ABI is present, there is no indication for an arteriogram. If an arterogram is obtained, the main arteries will be intact, and the muscle branches will taper. Anticoagulation does not treat compartment syndrome.

#### **REFERENCES (6)**

Feliciano DV, Cruse PA, Spjut-Patrinely V, et al. Fasciotomy after trauma to the extremities. Am J Surg 1988; 156:533-536.

Johansen K, Lynch K, Paun M, et al. Noninvasive vascular tests reliably exclude occult arterial trauma in injured extremities. *J Trauma* 1991; 31:515-522.

Whitesides TE, Heckman MM. Acute compartment syndrome: update on diagnosis and treatment. J Am Acad Orthop Surg 1996; 4:209-218.

RATIONALE (7)

Answer: D

Blunt aortic injury is the second most common cause of early death in blunt trauma patients after central nervous system injury. The majority of patients die at the scene, with 13 to 15% arriving at the hospital with signs of life. The remainder of patients will die within the first few days of hospitalization if this injury is not diagnosed and treated. The most common mechanism of blunt aortic injury appears to be from a motor vehicle collision, with frontal and lateral impacts occurring with equal frequency. Other common mechanisms include pedestrian versus motor vehicle accidents and falls.

The chest radiograph has been extensively studied as a screening test. A wide mediastinum has been the most frequently cited chest radiograph finding, which triggers additional workup for blunt aortic injury. A wide mediastinum may be defined as a measured width >8 cm. Mediastinal abnormalities on chest radiograph that are considered strongly suggestive of blunt aortic injury include an obscure or indistinct aortic knob, depression of the left main bronchus, deviation of a nasogastric tube, and opacification of the aortopulmonary window. Apical capping and widened peritracheal or perispinous stripes are also seen. It is possible for blunt aortic injury to occur in the face of a normal chest radiograph. Therefore, patients with significant deceleration mechanism should undergo a further screening test.

Angiography has been used as the "gold standard" to diagnose blunt aortic injury. It is the test to which all others are compared. CT scan of the chest appears to be a very useful diagnostic tool. Its use ranges from screening of all patients with blunt chest trauma to studying only those individuals with normal or low suspicion chest radiographs. A potential problem with CT scan is that it may delay time to angiography and, therefore, to a definitive diagnosis. This problem is resolved with

newer generation scanners, such as helical or spiral CT scanners. These machines are more sensitive and appear to have 100% negative predictive value. Once spiral CT scan is used, angiography may be reserved for patients with indeterminate scans.

Once the diagnosis of blunt aortic injury is made, most authors agree that prompt surgical repair is the best approach.

#### **REFERENCES (7)**

Fabian TC, Davis KA, Gavant ML, et al. Prospective study of blunt aortic injury: helical CT is diagnostic and antihypertensive therapy reduces rupture. *Ann Surg* 1998; 227:666-677.

Fabian TC, Richardson JD, Croce MA, et al. Prospective study of blunt aortic injury: multicenter trial of the American Association of the Surgery of Trauma. *J Trauma* 1997; 42:374-383.

Katyal D, McLellan BA, Brenneman FD, et al. Lateral impact motor vehicle collisions: significant cause of blunt traumatic rupture of the thoracic aorta. *J Trauma* 1997; 42:769-772.

Mirvis SE, Shanmuganathan K, Buell J, et al. Use of spiral computed tomography for the assessment of blunt trauma patients with potential aortic injury. *J Trauma* 1998; 45:922-930.

The EAST Practice Management Guidelines Work Group. Guidelines for the diagnosis and management of blunt aortic injury. Available at: www.east.org/tpg.html. Accessed June 11, 2007.

RATIONALE (8)

Answer: E

Posttraumatic empyema is a significant problem in both blunt and penetrating chest injuries. Potential etiologies include iatrogenic infection of the pleural space as during chest tube placement; direct infection resulting from penetrating injuries; secondary infection of the pleural space from associated intraabdominal organ injuries with diaphragm disruption; secondary infection of undrained or inadequately drained hemothorax; hematogenous or lymphatic spread of subdiaphragmatic infection and parapneumonic infection resulting from posttraumatic pneumonia; or pulmonary contusion.

Organisms responsible for infection vary according to mechanism of contamination. When related to chest tube insertion, empyema typically will be Gram-positive, *Staphylococcus aureus*, or Streptococcus species. Later contamination from pneumonic processes or other routes of spread may involve Gram-negative or mixed bacterial pathogen results.

One possible interventional use of prophylactic antibiotics in patients requiring tube thoracostomy is for traumatic hemothorax or pneumothorax. However, this terminology is a misnomer in trauma patients. By definition, prophylactic antibiotic regimens achieve a preinoculation serum and tissue drug concentration before bacterial contamination—an impossibility in a trauma patient. Therefore, antibiotic administration in the immediate postinjury period is more correctly considered presumptive therapy.

Multiple factors contribute to the development of posttraumatic empyema. In control groups, the incidence of empyema ranges between 0 to 18%. The administration of antibiotics for longer than 24 hours did not appear to significantly reduce the risk compared with a shorter duration although the numbers in each series are small.

#### **REFERENCES (8)**

Cant PJ, Smyth S, Smart DO. Antibiotic prophylaxis is indicated for chest stab wounds requiring closed tube thoracostomy. *Br J Surg* 1993; 80:464-466.

Demetriades D, Breckon V, Breckon C, et al. Antibiotic prophylaxis in penetrating injuries of the chest. *Ann R Coll Surg Engl* 1991; 73:348-351.

Oparah SS, Mandal AK. Penetrating stab wounds of the chest: experience with 200 consecutive cases. *J Trauma* 1976; 16:868-872.

The EAST Practice Management Guidelines Work Group. Practice management guidelines for prophylactic antibiotic use in tube thoracostomy for traumatic hemopneumothorax. Available at: www.east.org/tpg.html. Accessed June 11, 2007.

RATIONALE (9)

Answer: C

This patient's radiograph is consistent with traumatic rupture of the diaphragm secondary to blunt abdominal trauma. Although in this case the findings are obvious, radiographic findings are only diagnostic in 16-30% of patients with diaphragmatic injury. Radiographic findings include a very high diaphragmatic shadow, the presence of bowel in the chest, and nasogastric tube passing high into the left hemithorax. With blunt injury, the diaphragmatic hernia results from a burst injury of the diaphragm secondary to a rapid increase in abdominal pressure. The cause may be motor vehicle crash, falls, kicks, and crush injuries. Currently, motor vehicle crash from lateral impact results in this injury more frequently than frontal collisions. With blunt injuries, most diaphragmatic hernias occur on the left side (85%), followed by the right and bilateral injuries. Stomach, colon, omenteum, spleen, and small bowel can cause herniation into the chest. Cardiopulmonary compromise may occur from the abdominal contents effect on the chest cavity. Both venous return and vital capacity are reduced. Immediate surgical repair is recommended. CT scan is the best noninvasive diagnostic modality, but it cannot rule out subtle injury. Additional contrast studies can be obtained to help make the diagnosis. Failure to recognize a traumatic diaphragmatic hernia predisposes patients to the complication of bowel obstruction or strangulation in the future. In the ventilated patient, a diaphragmatic hernia may not be recognized until the patient is extubated.

#### **REFERENCES (9)**

- Asensio JA, Petrone P, Demetriades D. Injury to the diaphragm. In: Trauma, 5th ed. Moore EE, Feliciano DV, Mattox KL, eds. New York, NY: McGraw-Hill, 2004, 613-635.
- Ilgenfritz FM, Stewart DE. Blunt trauma of the diaphragm: a 15 county private hospital experience. Am Surg 1992; 58:334-339.
- Kearney PA, Rouhana SW, Burney RE. Blunt rupture of the diaphragm: mechanism, diagnosis, and treatment. *Ann Emerg Med* 1989;18:1326-1330.
- Reiff DA, Davis RP, MacLennan PA, et al. The association between body mass index and diaphragm injury among motor vehicle collision occupants. *J Trauma* 2004;57:1324-1328.
- Shapiro MJ, Heiberg E, Durham RM, et al. The unreliability of CT scans and initial chest radiographs in evaluating blunt trauma induced diaphragmatic rupture. *Clin Radiol* 1996; 51:27-30.
- Wiencek RG Jr., Wilson RF, Steiger Z. Acute injuries of the diaphragm: an analysis of 165 cases. J Thorac Cardiovasc Surg 1986; 92:989-993.

RATIONALE (10)

Answer: C

Severely injured trauma victims are at high risk of development of the multiple organ dysfunction syndrome or death. To maximize chances for survival, treatment priorities must focus on resuscitation from shock, defined as inadequate tissue oxygenation to meet requirements including appropriate fluid resuscitation and rapid hemostasis. Traditional markers of successful resuscitation, including restoration of normal blood pressure, heart rate and urine output, remain the standard-of-care per the Advanced Trauma Life Support Course. After normalizing these parameters, however, up to 85% of severely injured trauma patients will still have evidence of inadequate tissue oxygenation based on findings of ongoing metabolic acidosis or evidence of gastric mucosal ischemia. Recognition of this state and its rapid reversal are critical to minimize the risk of multiple organ dysfunction or death.

## **REFERENCES (10)**

- Abou-Khalil B, Scalea TM, Trooskin SZ, et al. Hemodynamic responses to shock in young trauma patients: need for invasive monitoring. *Crit Care Med* 1994; 22:633-639.
- Scalea TM, Maltz S, Yelon J, et al. Resuscitation of multiple trauma and head injury: Role of crystalloid fluids and inotropes. *Crit Care Med* 1994; 22:1610-1615.
- The EAST Practice Management Guidelines Work Group. Clinical practice guideline: Endpoints for resuscitation. Available at: www.east.org/tpg.html. Accessed June 11, 2007.
- Tisherman SA, Barie P, Bokhari F, et al. Clinical practice guideline: Endpoints of resuscitation. J Trauma 2004;57:898-912.

RATIONALE (11)

Answer: C

Elevated base deficit is not only predictive of mortality but of complications, such as the need for blood transfusions and organ failure, particularly acute respiratory distress syndrome. Elevated lactate and base deficit during the first 24 hours of admission has been associated with elevated IL-6 levels and with neutrophil CD11B expression, suggesting that inflammatory processes are involved in the relationship between severity of posttraumatic shock and later development of organ failure. A prospective study was done of 40 patients requiring operations for truncal injuries to identify factors to best determine which patients were at greatest risk for developing hemodynamic instability. These workers found that both base deficit and lactate levels corresponded with transfusion requirements, but mixed venous oxygen saturation did not.

#### **REFERENCES (11)**

- Eachempati SR, Robb T, Ivatury RR, et al. Factors associated with mortality in patients with penetrating abdominal vascular trauma. *J Surg Res* 2002; 108:222-226.
- Davis JW, Parks SN, Kaups KL, et al. Admission base deficit predicts transfusion requirements and risk of complications. *J Trauma* 1996; 41:769-774.
- Gattinoni L, Brazzi L, Pelosi P, et al. A trial of goal-oriented hemodynamic therapy in critically ill patients. N Engl J Med 1995; 333:1025-1032.
- Krishna G, Sleigh JW, Rahman H. Physiological predictors of death in exsanguinating trauma patients undergoing conventional trauma surgery. *Aust N Z J Surg* 1998; 68:826-829.
- The EAST Practice Management Guidelines Work Group. Clinical practice guideline: endpoints for resuscitation. Available at: www.east.org/tpg.html. Accessed June 11, 2007.
- Tisherman SA, Barie P, Bokhari F, et al. Clinical practice guideline: Endpoints of resuscitation. J Trauma 2004;57:898-912.

RATIONALE (12)

Answer: B

Immediate repair of blunt aortic injury may not be possible in all patients. These include patients who are unstable from intraabdominal injuries and require laparotomy or patients with severe closed head injury who require craniotomy. Another subset of patients is those who are elderly or have comorbidities that prohibit emergency thoracic surgery. These patients may be managed medically until other factors have resolved. Pharmacologic control of blood pressure and heart rate with  $\beta$ -blockers, possibly with nitroprusside, is extremely important when delayed or nonoperative management of blunt aortic injury is considered.

This patient requires immediate attention for his peritonitis. Medical management of intracranial pressure and control of blood pressure will be performed until abdominal problems are addressed. He should proceed to laparotomy rather than remaining in the emergency department. Treatment of an open fracture will require operation but at a later time.

#### **REFERENCES (12)**

- Borman KR, Aurbakken CM, Weigelt JA. Treatment priorities in combined blunt abdominal and aortic trauma. Am J Surg 1982; 144:728-732.
- Hemmila MR, Arbabi S, Rowe SA, et al. Delayed repair for blunt thoracic aortic injury: Is it really equivalent to early repair? *J Trauma* 2004;56:13-23.
- Hudson HM 2nd, Woodson J, Hirsch E. The management of traumatic aortic tear in the multiply-injured patient. *Ann Vasc Surg* 1991; 5:445-448.
- Maggisano R, Nathens A, Alexandrova NA, et al. Traumatic rupture of the thoracic aorta: Should one always operate immediately? *Ann Vasc Surg* 1995; 9:44-52.
- The EAST Practice Management Guidelines Work Group. Guidelines for the diagnosis and management of blunt aortic injury. Available at: www.east.org/tpg.html. Accessed June 11, 2007.

RATIONALE (13)

Answer: E

This patient has received massive transfusion of cold blood products and is hypothermic. In order to correct the coagulopathy, the temperature must be normalized. There are many methods to accomplish this goal, including increasing the ambient temperature and warming all IV fluids and blood products. Fluids can be given through a blood warmer or through a rapid infusion device with a warming element. The temperature on the ventilator can be increased and the bladder or nasogastric tube can be irrigated with warm fluids. A chest drainage system can warm patients across the pleura with heated fluid exchange. In severe cases of hypothermia, patients have been rewarmed with cardiopulmonary bypass. During the rewarming process, fluid losses continue and must be replaced. The coaugulopathy cannot be reversed with clotting factors alone. Neither reoperation nor angiographic embolization have a role until the patient's temperature is normalized.

#### **REFERENCES (13)**

- Gentilello LM, Jurkovich GJ, Stark MS, et al. Is hypothermia in the victim of major trauma protective or harmful? A randomized, prospective study. *Ann Surg* 1997; 226:439-449.
- Hess JR, Lawson JH. The coagulopathy of trauma versus disseminated intravascular coagulation. J Trauma 2006;60:S12-S19.
- Holcomb JB, Jenkins D, Rhee P, et al. Damage control resuscitation: Directly addressing the early coagulopathy of trauma. J Trauma 2007;62:307-310.
- Johnston TD, Chen Y, Reed RL 2nd. Functional equivalence of hypothermia to specific clotting factor deficiencies. *J Trauma* 1994; 37:413-417.
- Reed RL 2nd, Bracey AW Jr., Hudson JD, et al. Hypothermia and blood coagulation: disassociation between enzyme activity and clotting levels. *Circ Shock* 1990; 32:141-152.
- Valeri CR, Feingold H, Cassidy G, et al. Hypothermia-induced reversible platelet dysfunction. *Ann Surg* 1987; 205:175-181.

RATIONALE (14)

Answer: E

An early report by Margolies and colleagues in 1972 on the use of angiography in the management of pelvic fracture-associated hemorrhage represented a fundamental change in the approach to this important problem. Inadequacies of operative exposure and ligation of bleeding sources in the pelvis are noted by multiple authors. The addition of selective angiography and embolization to the armamentarium represented a new modality in the treatment of arterial bleeding secondary to pelvic fracture and rapidly became widespread. As the resolution power of CT scanning improved, it developed the ability to identify arterial extravasation of IV contrast. The recognition of this fact in the pelvis moved angiography out of a diagnostic role to a more strictly therapeutic role. Multiple attempts to identify fracture patterns predictive of arterial injuries in the pelvis have been unsuccessful.

#### **REFERENCES (14)**

- Kimbrell BJ, Velmahos GC, Chan LS, et al. Angiographic embolization for pelvic fractures in older patients. *Arch Surg* 2004;139:728-733.
- Margolies MN, Ring EJ, Waltman AC, et al. Arteriography in the management of hemorrhage from pelvic fractures. N Engl J Med 1972; 287:317-321.
- Poole GV, Ward EF. Causes of mortality in patients with pelvic fractures. *Orthopedics* 1994; 17:691-696. Rothenberger DA, Fischer RP, Strate RG, et al. The mortality associated with pelvic fractures. *Surgery* 1978; 84:356-361.
- The EAST Practice Management Guidelines Work Group. Clinical practice guideline: practice management guidelines for hemorrhage in pelvic fracture. Available at: www.east.org/tpg.html. Accessed June 11, 2007.
- Velmahos GC, Toutouzas KG, Vassiliu P, et al. A prospective study on the safety and efficacy of angiographic embolization for pelvic and visceral injuries. *J Trauma* 2002;53:303-308.

RATIONALE (15)

Answer: C

Indications for laparotomy in the face of pelvic fracture and hypotension remain the same indications for laparotomy in the absence of pelvic fracture: significant intraabdominal hemorrhage and perforation of the gastrointestinal tract. Involuntary guarding, suggesting peritonitis or a large perihepatic fluid collection on abdominal ultrasound, are consistent with a significant intraabdominal process and are sufficient indications for operation. A large retroperitoneal fluid collection on CT scan of the abdomen and pelvis is inconsistent with a significant intraperitoneal, intraabdominal process. Hypotension after successful arterial embolization in the pelvis and placement of an external fixation device may reflect ongoing volume requirements to address venous bleeding in the retroperitoneum. In the absence of finding specific to the peritoneal cavity, exploratory laparotomy is not warranted.

#### **REFERENCES (15)**

- Evers BM, Cryer HM, Miller FB. Pelvic fracture hemorrhage: priorities in management. *Arch Surg* 1989; 124:422-424.
- Flint L, Babikian G, Anders M, et al. Definitive control of mortality from severe pelvic fracture. *Ann Surg* 1990; 211:703-707.
- Moreno C, Moore EE, Rosenberger A, et al. Hemorrhage associated with major pelvic fracture: a multispecialty challenge. *J Trauma* 1986; 26:987-994.
- Panetta T, Sclafani SJ, Goldstein AS, et al. Percutaneous transcatheter embolization for massive bleeding from pelvic fractures. *J Trauma* 1985; 25:1021-1029.
- The EAST Practice Management Guidelines Work Group. Clinical practice guideline: practice management guidelines for hemorrhage in pelvic fracture. Available at: www.east.org/tpg.html. Accessed June 11, 2007.

RATIONALE (16)

Answer: B

The widened mediastinum on this radiograph is suspicious for traumatic rupture of the aorta (TRA). Control of blood pressure to prevent hypertension is essential and will prevent stress on the wall of the pseudoaneurysm and help prevent rupture. This is treated similar to a dissection in that both HR and BP must be controlled. Either labatolol or a combination of a  $\beta$ -blocker and nitropruside are used. Nitropruside alone is insufficient because of the resulting tachycardia. Controlling the BP and HR allows time for adequate studies and preparation for operative repair of this injury. On occasion, medical management allows prevention of hemorrhage, while other injuries are addressed prior to aortic repair.

Most (80-90%) patients with TRA die at the scene, because the injury is typically through all layers of the aorta, causing immediate uncontrolled hemorrhage. The 10-20% of patients with TRA who reach the hospital alive have intact adventitia, and 50% of these patients die within 48 hours if not treated effectively. Patients presenting with acute abdominal hemorrhage should have this problem addressed *prior* to evaluation and repair of aortic injury. In the more stable patient, a trauma CT scan that includes chest and abdominal views can be obtained. CT scan is becoming the key diagnostic test for TRA. Bleeding from the mediastinal veins usually causes the mediastinal widening. The widened mediastinium is the most common abnormality seen on chest radiographs, but it is not specific for TRA. The most common site of injury to the aorta occurs just distal to the left subclavian artery at the aortic isthmus.

#### **REFERENCES (16)**

- Demetriades D, Gomez H, Velmahos GC, et al. Routine helical computed tomographic evaluation of the mediastinum in high-risk blunt trauma patients. *Arch Surg* 1998; 133:1084-1088.
- Dyer DS, Moore EE, Ilke DN, et al. Thoracic aortic injury: How predictive is mechanism and is chest computed tomography a reliable screening tool? A prospective study of 1,561 patients. *J Trauma* 2000; 48:673-683.
- Fabian TC, Davis KA, Gavant ML, et al. Prospective study of blunt aortic injury: helical CT is diagnostic and antihypertensive therapy reduces rupture. *Ann Surg* 1998; 227:666-677.
- Hemmila MR, Arbabi S, Rowe SA, et al. Delayed repair for blunt thoracic aortic injury: Is it really equivalent to early repair? *J Trauma* 2004; 56:13-23.
- Pretre R, Chilcott M. Blunt trauma to the heart and great vessels. N Engl J Med 1997; 336:626-632.

RATIONALE (17)

Answer: C

Inhalation injury is a consistent cause of increased mortality in burn victims. Upper airway injury is due to direct heat exposure but initial mortality is due to asphyxia. Laryngeal reflexes protect the lung from direct thermal injury. Lower airway injury is predominantly due to chemical pneumonitis from products of combustion carried to the lung on particles of soot.

Chest radiograph and arterial blood gas findings are inadequate for early diagnosis of inhalation injury, which evolves over time. Parenchymal pulmonary dysfunction is frequently limited for 24 to 72 hours. Classic components in presentation include a history of burn injury occurring in a closed space where inhalation of products of combustion cannot be avoided and physical examination revealing soot or burn injury about the face and particularly the nose and mouth. Pneumonia is the major late complication of inhalation injury and incrementally adds to mortality associated with this problem.

#### **REFERENCES (17)**

- American Burn Association. Practice guidelines for burn care. J Burn Care Rehabil Supplement 2001; 22:1S-69S.
- Miller SF, Bessey PQ, Schurr MJ, et al. National Burn Repository 2005: A ten-year review. *J Burn Care Res* 2006;27:411-436.
- Ryan CM, Schoenfeld DA, Thorpe WP, et al. Objective estimates of the probability of death from burn injuries. *N Engl J Med* 1998; 338:362-366.
- Shirani KZ, Pruitt BA Jr., Mason AD Jr. The influence of inhalation injury and pneumonia on burn mortality. *Ann Surg* 1987; 205:82-87.
- Smith DL, Cairns BA, Ramadan F, et al. Effect of inhalation injury, burn size, and age on mortality: a study of 1447 consecutive burn patients. *J Trauma* 1994; 37:655-659.

RATIONALE (18)

Answer: A

Carbon monoxide poisoning is a serious health problem resulting in approximately 40,000 annual visits to emergency departments in the United States. With thermal injury, it may be a significant source of additional morbidity. Cognitive sequelae may occur immediately after exposure and may persist or may be delayed in presentation. In general, neurologic changes occur within 20 days after carbon monoxide exposure. Standard treatment for acute carbon monoxide intoxication is 100% normobaric oxygen. Hyperbaric oxygen therapy is sometimes recommended for patients with acute carbon monoxide poisoning, particularly if they have lost consciousness or have high carboxyhemoglobin levels. Reported advantages to the administration of hyperbaric oxygen with carbon monoxide exposure include increased dissolved oxygen content in the blood and accelerated elimination of carbon monoxide. Potential benefits include prevention of lipid peroxidation in the central nervous system and preservation of adenosine triphosphate levels in tissues exposed to carbon monoxide. Recent work with patients experiencing significant carbon monoxide exposure in the absence of significant thermal injury suggests that neurologic outcome measured at 6 weeks and 12 months after acute exposure to carbon monoxide will improve with acute hyperbaric oxygen administration. Notably, the patient sustaining significant thermal cutaneous injury is not represented in these studies. Optimal management of carbon monoxide exposure in the setting of significant thermal cutaneous injury remains unclear, and challenges of early administration of hyperbaric oxygen in the setting of multisystem trauma remain significant.

Studies describing necrotizing infections and chronic refractory osteomyelitis report inconsistent results with hyperbaric oxygen administration as an adjunctive therapy to standard wound care. Only one case series in a recent review found hyperbaric oxygen to be beneficial. No confirming studies were available. Consistent criteria for hyperbaric oxygen use are unavailable in soft tissue infections and tissue ischemia. Even the optimal method for measuring tissue oxygenation has not been determined.

#### **REFERENCES (18)**

Ernst A, Zibrak JD. Carbon monoxide poisoning. N Engl J Med 1998; 339:1603-1608. Tibbles PM, Edelsberg JS. Hyperbaric oxygen therapy. N Engl J Med 1996; 334:1642-1648. Wang C, Schwaitzberg S, Berliner E, et al. Hyperbaric oxygen for treating wounds: a systematic review of the literature. Arch Surg 2003; 138:272-280.

Weaver LK, Hopkins RO, Chan KJ, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med 2002; 347:1057-1067. RATIONALE (19)

Answer: D

Probably the most popular resuscitation approach utilizes a modified Parkland formula giving 4 mL/kg/% total body surface area burn of fluid (lactated Ringers) with half of the required 24-hour volume given in the first 8 hours. After this initial fluid prescription, resuscitation is titrated to maintain urine output of 30-50 mL/h in the adult and adequate vital signs.

The existence of mathematical formulas to guide burn resuscitation leads many to the erroneous conclusion that burn resuscitation is consistent and predictable. In general, resuscitation amounts have been reported to exceed formula predictions, even with modest therapeutic goals. Recent reviews of resuscitation experience suggest that attempts to titrate resuscitation with pulmonary artery catheters lead to fluid administration in excess of that provided by other prescriptive approaches without improvement in metabolic outcome. The presence of inhalation injury, particularly if mechanical ventilation is required, also will increase the amount of fluid required.

Where supplemental colloids are employed, these are typically used during the second 24 hours of burn resuscitation. Colloid administration is an option in elderly patients and patients with large burns (>50% total body surface area).

#### **REFERENCES (19)**

- American Burn Association. Practice guidelines for burn care. J Burn Care Rehabil Supplement 2001; 22:1S-69S.
- Cancio LC, Chavez S, Alvarado-Ortega M, et al. Predicting increased fluid requirements during the resuscitation of thermally injured patients. *J Trauma* 2004; 56:404-414.
- Holm C, Mayr M, Tegeler J, et al. A clinical randomized study on the effects of invasive monitoring on burn shock resuscitation. *Burns* 2004; 30:798-807.
- Navar PD, Saffle JR, Warden GD. Effect of inhalation injury on fluid resuscitation requirements after thermal injury. Am J Surg 1985; 150:716-720.
- Saffle JR. The phenomenon of "fluid creep" in acute burn resuscitation. *J Burn Care Res* 2007;28:382-395.

RATIONALE (20)

Answer: B

In head injured patients, significant secondary brain injury results from systemic hypotension and hypoxemia. An English study revealed that 44% of victims of traumatic brain injury were hypoxemic in the field or ambulance, and episodes of hypotension are also extremely common. Deleterious effects of hypoxemia and hypotension on the outcome of severe head injury have also been demonstrated from the large Traumatic Coma Data Bank. In this dataset, hypoxemia and hypotension each occurred in over one third of severe head injury patients. Fluid administration, by raising mean arterial blood pressure improves cerebral perfusion pressure. Hypertonic saline solution and mannitol have been recommended, in addition to resuscitation fluids, as adjuncts to support cerebral perfusion pressure (mean arterial pressure minus cerebral perfusion pressure) >70 mm Hg. The target of resuscitation is cerebral perfusion pressure, not mean arterial blood pressure. Mannitol is avoided in the hypotensive patient.

In patients with severe head injury, major predictors of outcome are the severity of the primary cerebral insult, age, initial Glasgow Coma Scale Score, incidence of hypoxia and hypotension, and other secondary insults. Secondary insults influence blood pressure and oxygenation. Multiple injured patients did not have higher occurrence of secondary brain insults, and no worse outcome, compared with patients with isolated severe head injury if appropriate cerebral resuscitation, is provided.

Patients with a Glasgow coma scale score <9 typically require intubation, as they are at risk for inability to maintain their airway, despite supplemental oxygen. The same is true for hypoxemia.

#### **REFERENCES (20)**

Gennarelli TA, Champion HR, Sacco WU, et al. Mortality of patients with head injury and extracranial injury treated in trauma centers. *J Trauma* 1989; 29:1193-1201.

Gennarelli TA, Spielman GM, Langfitt TW, et al. Influence of the type of intracranial lesion on outcome from severe head injury. *J Neurosurg* 1982; 56:26-32.

Marik PE, Varon J, Trask T. Management of head trauma. Chest 2002; 122:699-711.

Miller JD, Becker DP. Secondary insults to the injured brain. JR Coll Surg Edinb 1982; 27:292-298.

Sarrafzadeh AS, Peltonen EE, Kaisers U, et al. Secondary insults in severe head injury. Do multiply injured patients do worse? Crit Care Med 2001; 29:1116-1123.

The Brain Trauma Foundation. The Joint Section on Neurotrauma and Critical Care: resuscitation of blood pressure and oxygenation. *J Neurotrauma* 2000; 17:471-478.

RATIONALE (21)

Answer: D

Replace 140% of resting metabolism expenditure in nonparalyzed patients and 100% of resting metabolism expenditure in paralyzed patients using enteral or parenteral formulas containing at least 15% of calories as protein by day 7 after injury. Two class I studies examined the effect of the extent of nutritional replacement on patient outcome. These showed that, with nearly equivalent quantities of feeding, the mode of administration (parenteral or enteral) had no effect on neurologic outcome.

There are a number of potential advantages to enteral feeding, including less risk of hyperglycemia, lower theoretical risk of infection, and reduced cost. Clearly, patients receiving parenteral nutrition require careful monitoring for hyperglycemia and will receive more insulin therapy than patients receiving enteral nutrition.

## **REFERENCES (21)**

Ott L, Young B, Phillips R, et al. Altered gastric emptying in the head-injured patient: relationship to feeding intolerance. *J Neurosurg* 1991; 74:738-742.

Robertson CS, Goodman JC, Narayan RK, et al. The effect of glucose administration on carbohydrate metabolism after head injury. *J Neurosurg* 1991; 74:43-50.

Suchner U, Senftleben U, Eckart T, et al. Enteral versus parenteral nutrition: effects on gastrointestinal function and metabolism. *Nutrition* 1996; 12:13-22.

The Brain Trauma Foundation. The Joint Section on Neurotrauma and Critical Care: nutrition. J Neurotrauma 2000; 17:539-547.

RATIONALE (22)

Answer: C

One of the most important principles in management of traumatic brain injury is avoidance of secondary brain injury. As with all trauma cases, the first step in management is assessment of airway, breathing and circulation (ABCs) to ensure there is not an immediate threat of secondary brain injury. After the ABCs, the disability should then be rapidly evaluated with the Glasgow Coma Score (GCS). Patients with severe head injury are at especially high risk for hypoxic events after the primary brain injury. This may be the result of respiratory depression or inability to maintain a patent airway. Prehospital literature suggests that severely brain-injured patients often suffer hypoxic events during transport after primary brain injury. For this reason, it is recommended that patients with a GCS  $\leq$ 8 require intubation and mechanical ventilation. In the clinical scenario provided, this patient's GCS is 7, and an artificial airway should be established immediately. Transporting the patient to the radiology department for a CT scan without first initiating mechanical ventilation would put the patient at high-risk for secondary (hypoxic) brain injury.

Because the patient's intracranial pressure may be severely elevated, it is imperative that the intubation be performed in such a way as to avoid airway stimulation and gagging, which could transiently increase intracranial pressure further.

Because hyperventilation may cause cerebral vasoconstriction and secondary brain injury, hyperventilation is no longer routinely recommended in cases of suspected elevated intracranial pressure. However, short-term hyperventilation is still indicated for transient lowering of the intracranial pressure in cerebral herniation syndrome.

#### **REFERENCES (22)**

Brain Trauma Foundation. Joint Section on Neurotrauma and Critical Care: hyperventilation. *J Neurotrauma* 2000; 17:513-520.

Marik PE, Varon J, Trask T. Management of head trauma. Chest 2002; 122:699-711.

The Brain Trauma Foundation. The Joint Section on Neurotrauma and Critical Care: initial management. *J Neurotrauma* 2000; 17:463-469.

Stocchetti N, Furlan A, Volta F. Hypoxemia and arterial hypotension at the accident scene in head injury. *J Trauma* 1996; 40:764-767.

Vincent JL, Berre J. Primer on medical management of severe brain injury. *Crit Care Med* 2005; 33:1392-1399.

RATIONALE (23)

Answer: C

Cerebral perfusion pressure (mean arterial pressure minus intracranial pressure) should be maintained at a minimum of 70 mm Hg. It has been argued that hypertensive therapy needed in some head-injured patients to maintain an adequate cerebral perfusion pressure can cause an increase in intracranial pressure and poor outcome. The effect of artificial blood pressure elevation on intracranial pressure and cerebral blood flow has been systematically studied in patients with severe traumatic brain injury. Elevation of mean arterial blood pressure from approximately 90 mm Hg to in excess of 120 mm Hg caused significant decrease in intracranial pressure in a number of patients. Intracranial pressure usually changes very little when blood pressure is increased by as much as 30 mm Hg in head injured patients. In one study, progressive decrease in cerebral perfusion pressure from 80 mm Hg increased mortality by 20% for each 10 mm Hg fall. When cerebral perfusion pressure was <60 mm Hg, mortality increased more rapidly.

#### **REFERENCES (23)**

- Bouma GJ, Muizelaar JP. Relationship between cardiac output and cerebral blood flow in patients with intact and with impaired autoregulation. *J Neurosurg* 1990; 73:368-374.
- Bruce DA, Langfitt TW, Miller JD, et al. Regional cerebral blood flow, intracranial pressure, and brain metabolism in comatose patients. *J Neurosurg* 1973; 38:131-144.
- McGraw CP. A Cerebral perfusion pressure greater than 80 mm Hg is more beneficial. In: Intracranial pressure VII. Hoff JT, Betz AL, eds. Berlin: Springer-Verlag; 1989, 839-841.
- Rosner MJ, Rosner SD, Johnson AH. Cerebral perfusion pressure: management protocol and clinical results. *J Neurosurg* 1995; 83:949-962.
- The Brain Trauma Foundation. The Joint Section on Neurotrauma and Critical Care: guidelines for cerebral perfusion pressure. *J Neurotrauma* 2000; 17:507-511.

RATIONALE (24)

Answer: E

The appearance of intracerebral (intraparenchymal) hematomas on CT scanning may be delayed as much as 24 hours after the primary injury. Regardless of initial CT scan results, a patient with rapid neurologic deterioration should have repeat imaging immediately after ABCs are secured.

There is no role for glucocorticoids in the management of traumatic brain injury. Prophylactic anticonvulsant therapy is recommended for 7 days in the routine care of all severely brain-injured patients.

Intracranial hypertension (intracranial pressure  $\geq 20$ ) is present in more than half of patients with Glasgow Coma Score (GCS)  $\leq 8$ . Therefore, an intracranial pressure monitor should be placed for the patient in this clinical scenario because the Glasgow Coma Score is 7. An intraventricular catheter is preferred, because it would allow for drainage of cerebrospinal fluid to decrease intracranial pressure.

Diffuse axonal injury is caused by shearing forces in the brainstem. Axons are torn in the reticular activating system. The injury may evolve as edema worsens and may not manifest until hours after the primary injury. If patients survive, the outcome is frequently prolonged unconsciousness or persistent vegetative state. The diagnostic study of choice for diffuse axonal injury is magnetic resonance imaging.

#### **REFERENCES (24)**

Fukamachi A, Kohno K, Nagaseki Y, et al. The incidence of delayed traumatic intracerebral hematoma with extradural hemorrhages. *J Trauma* 1985; 25:145-149.

Marik PE, Varon J, Trask T. Management of head trauma. Chest 2002; 122:699-711.

Soloniuk D, Pitts LH, Lovely M, et al. Traumatic intracerebral hematomas: timing of appearance and indications for operative removal. *J Trauma* 1986; 26:787-794.

The Brain Trauma Foundation. The Joint Section on Neurotrauma and Critical Care: indication for intracranial pressure monitoring. *J Neurotrauma* 2000; 17:479-491.

The Brain Trauma Foundation. The Joint Section on Neurotrauma and Critical Care: role of antiseizure prophylaxis following head injury. *J Neurotrauma* 2000; 17:549-553.

The Brain Trauma Foundation. The Joint Section on Neurotrauma and Critical Care: role of steroids. J Neurotrauma 2000; 17:531-535.

Vincent JL, Berre J. Primer on medical management of severe brain injury. *Crit Care Med* 2005; 33:1392-1399.

RATIONALE (25)

Answer: A

Any break in the skin allows bacteria to colonize a wound within hours. Infection occurs when bacteria subsequently invades surrounding viable tissue with a resulting host response. Bacteria with this capability are defined as pathogens but even minimally virulent bacteria may establish infection when local or systemic host defenses are impaired. Local trauma with tissue edema, hematoma, ischemic tissue, or a foreign body increases the risk of infection. Systemic conditions associated with more virulent soft tissue infection include diabetes, malignancy, malnutrition, major soft tissue disruption, advanced age, atherosclerosis, and use of steroids.

The role of immediate antibiotic administration is debated. Clearly, a wound such as this requires operative debridement. Operative debridement rather than antibiotic administration is the most important aspect of immediate therapy.

This patient has significant exposure to dirt and must be considered to have a tetanus prone wound. Administration of tetanus immune globulin is indicated for any patient with such a wound if tetanus immunization is incomplete or immunization status is unknown.

#### **REFERENCES (25)**

Ahrenholz DH. Necrotizing fasciitis and other soft tissue infections. In: *Irwin and Rippe's Intensive Care Medicine*. 5th ed. Irwin RS, Rippe JM, eds. Philadelphia, PA: Lippincott Williams & Wilkins; 2003, 1709-1716.

McHenry CR, Piotrowski JJ, Petrinic D, et al. Determinants of mortality for necrotizing soft-tissue infections. *Ann Surg* 1995; 221:558-565.

Stewart RM, Myers JG, Dent DL. Wounds, bites, and stings. In: *Trauma, Fifth Edition*. Moore EE, Feliciano DV, Mattox KL, eds. New York, NY: McGraw Hill; 2004, 1059-1079.

RATIONALE (26)

Answer: D

This patient has a clear pelvic abscess as indicated by the rim enhancing lesion seen on the more recent CT scan images. A lesion such as this should be immediately evacuated either in interventional radiology or the operating room. The initial procedure of choice is percutaneous drainage in the interventional radiology suite. The vast majority of abscess fluid collections should be treated in this way. Antibiotic coverage should include management of Gram-negative and anaerobic organisms. The possibility of gastrointestinal tract injury is suggested by the history of a bloody bowel movement and indicates a potential source for organisms in the abscess fluid. Antibiotics in this case are warranted for 5 days. If additional antibiotic coverage is desirable, additional imaging or surgery should be considered to justify further drug use. A CT scan image with a drain in place is shown below.



## REFERENCES (26)

Christou NV, Barie PS, Dellinger EP, et al. Surgical Infection Society intra-abdominal infection study. Prospective evaluation of management techniques and outcome. *Arch Surg* 1993;128:193-199. Mazuski JE, Sawyer RG, Nathens AB, et al. The Surgical Infection Society guidelines on antimicrobial therapy for intraabdominal infections: an executive summary. *Surg Infect* 2002; 3:161-173.

Solomkin JS, Mazuski JE, Baron EJ, et al. Guidelines for the selection of anti-infective agents for complicated intraabdominal infections. *Clin Infect Dis* 2003; 37:997-1005.

RATIONALE (27)

Answer: C

A Glasgow Coma Scale score  $\leq 8$  is associated with a significant chance of intracranial pressure  $\geq 20$  cm  $H_2O$  and is typically an indication for intracranial pressure monitoring. The utility of blood transfusions in trauma has seen extensive evaluation. Blood transfusions are an independent predictor of multiorgan failure, particularly when blood older than 2 weeks is administered. ICU patients can be safely managed with hemoglobin levels in the range of 7 gm/dL. Mechanisms by which old blood potentiates organ failure include the proinflammatory nature of plasma and lipid factors, which accumulate during the storage period and reduce stability of stored red blood cells to traverse the microcirculation resulting in microcirculatory dysfunction.

The Traumatic Coma Data Bank demonstrates that hypotension and hypoxia (systolic blood pressure <90 mm Hg and Pao<sub>2</sub> <60 mm Hg is associated with increased morbidity and mortality in the setting of traumatic brain injury. Due to the low Glasgow Coma Scale score in this patient, intubation and intracranial pressure monitoring are appropriate. Where intracranial pressure monitoring is available, vasoactive therapy, including fluids and vasoactive drugs, if necessary, should be given to maintain cerebral perfusion pressure (mean arterial pressure minus intracranial pressure) of at least 60 mm Hg.

There is some evidence that patients with significant head trauma may be harmed by early-operative fracture stabilization due to a higher prevalence of acute respiratory distress syndrome and secondary brain injury. Nonetheless, most evidence supports proceeding to the operating room for early fracture fixation as an effective method of reducing organ failure in patients with long bone fractures.

#### **REFERENCES (27)**

- Brain Trauma Foundation. Joint Section on Neurotrauma and Critical Care Guidelines: resuscitation of blood pressure and oxygenation. *J Neurotrauma* 2000; 17:471-478.
- Deitch EA, Dayal SD. Intensive care unit management of the trauma patient. Crit Care Med 2006; 34:2294-2301.
- Hebert PC, Wells G, Blajchman MA, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. N Engl J Med 1999; 340:409-417.
- Juul N, Morris GF, Marshall SB, et al. Intracranial hypertension and cerebral
- perfusion pressure: influence on neurological deterioration and outcome in severe head injury—the Executive Committee on the International Selfotel Trial. *J Neurosurg* 2000; 92:1-6.
- Moore FA, Moore EE, Sauaia A. Blood transfusion: an independent risk factor for postinjury multiple organ failure. *Arch Surg* 1997; 132:620-625.

RATIONALE (28)

Answer: D

Along with burn size >50% total body surface area and age >50-55 years, inhalation injury is an independent factor predicting poor outcome following thermal trauma. A number of respiratory strategies have been evaluated. While some recent work suggests improved outcomes in inhalation injury with early tracheostomy, provision of a surgical airway early in the course of burn patients remains controversial. While clinicians agree that if tracheostomy is employed, the percutaneous route is preferred. This approach must be taken with caution in patients with severe head and neck burns (common in inhalation injury). There are a variety of small, typically single center, studies supporting the efficacy of high-frequency oscillation and high-frequency percussive ventilation in burn-injured patients with refractory hypoxemia. These strategies have not been widely employed, despite theoretical attractiveness. Low tidal volume, lung protective ventilation with positive end expiratory pressure used as recommended by the recent ARDSNet trial remains the standard of care for the burn-injured patient. Tracheostomy may be needed later in the hospital course, if extubation cannot be accomplished, but specific timing for this intervention has not been conclusively established.

#### **REFERENCES (28)**

Ipaktchi K, Arbabi S. Advances in burn critical care. *Crit Care Med* 2006; 34(suppl):S239-S244. Pelosi P, Severgnini P. Tracheostomy must be individualized. *Crit Care* 2004; 8:322-324. Saffle JR, Morris SE, Edelman L. Early tracheostomy does not improve outcome in burn patients. *J Burn Care Rehabil* 2002; 23:431-438.

RATIONALE (29)

Answer: A

Fat embolism occurs commonly during orthopedic surgery. The key triad of symptoms includes neurologic changes, respiratory distress, and petechial rash (see figure below). Surgical procedures most commonly associated with this complication are total hip arthorplasty, total knee arthroplasty, and femur intramedullary rod placement. Pathogenesis is poorly understood, but biochemical and mechanical phenomenon are postulated.

Diagnosis is based on a variety of major and minor criteria. Major diagnostic criteria are respiratory distress, neurologic status changes, and petechial rash. Minor criteria include pyrexia, renal insufficiency, elevated bilirubin, and tachycardia. Signs and symptoms may present immediately or within 24 to 48 hours. Respiratory distress is the most frequent finding, and neurologic changes occur in 86% of patients. Petechial rash is pathonomonic for fat embolism syndrome but occurs in only 40% of individuals.

Treatment is supportive and mortality ranges from 5 to 33%. Heparin and steroids have not shown efficacy. Bronchoscopy will likely demonstrate lipid-laden macrophages. This test is unnecessary, however, given the above diagnostic information.

#### The Schonfeld Criteria for the Diagnosis of Fat Embolism Syndrome

Criteria	Points*
Petechiae	5
Chest radiograph changes	4
Hypoxemia	3
Fever	1
Tachycardia	1
Tachypnea	1
Confusion	1

<sup>\*</sup>Fat embolism syndrome defined by a score of >5.

Crit Care Med 2006; 34[Suppl]:S191-S199

#### **REFRENCES (29)**

Nazon D, Abergel G, Hatem CM. Critical care in orthopedic and spine surgery. Crit Care Clin 2003; 19:33-53.

Ries MD, Lynch F, Rauscher LA, et al. Pulmonary function during and after total hip replacement: findings in patients who have insertion of a femoral component with and without cement. *J Bone Joint Surg Am* 1993; 75:581-587.

Schonfeld SA, Ploysongsang Y, DiLisio R, et al. Fat embolism prophylaxis with corticosteroids. A prospective study in high-risk patients. *Ann Intern Med* 1983; 99:438-443.

Taylor JM, Gropper MA. Critical care challenges in orthopedic surgery patients. *Crit Care Med* 2006;34(Suppl):S191-S199.

RATIONALE (30)

Answer: D

This patient has class II hemorrhagic shock by Advanced Trauma Life Support criteria. The classifications I through IV are shown below (see Table 1). Class IV shock is not a subtle clinical presentation, as the patient has lost more than 40% of blood volume and has profound hypotension, altered mental status, acidosis, and anuria. Class I shock, on the other hand, may have very subtle clinical signs, where a patient can lose up to 15% of blood volume and be very well compensated, with nearly no derangements of global hemodynamic parameters, including BP and HR. Class II hemorrhagic shock is characterized by tachycardia in the setting of a blood loss in the range of 15 to 30% of blood volume. Pulse pressure is typically decreased. A pulse pressure of 23 mm Hg in this patient scenario is in abnormal range. Slightly diminished urine output and increased respiratory

rate are observed with mild anxiousness. Crystalloid resuscitation should be the mainstay of therapy for this degree of shock. The hallmark of class III hemorrhagic shock is hypotension, and it is also characterized by oliguria and altered mental status. Class III hemorrhagic shock represents the threshold to initiate blood products in the initial resuscitation. In this patient scenario, if the patient's clinical condition would deteriorate and manifest these signs of hypoperfusion, blood would be indicated. However, at this point, crystalloid therapy should be administered and would likely be sufficient, if operative control of the bleeding can be achieved promptly. Indiscriminate use of blood transfusion is not recommended, as the potential deleterious effects of blood transfusion are becoming better appreciated, including immunomodulating effects and increased risk of nosocomial infection.

Table 1—Estimated Fluid and Blood Losses¹
Based on Patient's Initial Presentation

	CLASS I	CLASS II	CLASS III	CLASS IV
Blood loss (mL)	Up to 750	750-1500	1500-2000	>2000
Blood loss (% blood volume)	Up to 15%	15%-30%	30%-40%	>40%
Pulse rate	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (num Hg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14-20	20-30	30-40	225
Urine output (mL/hr)	>30	20-30	5-15	>35 Negligible
CNS/Mental status	Slightly anxious	Mildly	Anxious,	Confused, lethargic
Fluid replacement (3:1 rule)	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

## **REFERENCE (30)**

Advanced trauma life support manual. 7th ed. Chicago, IL: American College of Surgeons Committee on Trauma, American College of Surgeons, 2004

RATIONALE (31)

Answer: C

The Focused Assessment Sonography in Trauma (FAST) examination is a systematic application of ultrasound in order to detect the presence of free fluid in the abdomen (presumably hemoperitoneum) in cases of blunt trauma. The FAST examination also evaluates the pericardial sac, in addition to the hepatorenal fossa, splenorenal fossa, and pelvis. In this patient scenario, the patient had cardiac tamponade (in addition to hemoperitoneum), and focusing solely on an intraabdominal source of hemorrhagic shock would have missed the pericardial process leading to obstructive shock. However, the patient also had hemoperitoneum from solid organ injury contributing to the shock profile. In the hands of a skilled operator, the FAST examination could have identified the immediate need for drainage of pericardial fluid in the trauma bay. The rest of the FAST examination would have identified hemoperitoneum and a need for laparotomy, as well. If the FAST examination identified no pericardial process but free fluid in the abdomen, then the correct decision for the hypotensive patient would have been to proceed immediately to exploratory laparotomy for control of the bleeding. Obtaining a CT scan would have been a poor decision, as the patient is in shock, and a rapid bedside diagnostic evaluation is needed. A CT scan is the diagnostic test of choice for patients with similar blunt injuries who are not markedly hypotensive. As proper chest tube placement has already been confirmed, a repeat chest radiograph is not necessary.

#### **REFERENCE (31)**

Advanced trauma life support manual. 7th ed. Chicago, IL: American College of Surgeons Committee on Trauma, American College of Surgeons, 2004.

RATIONALE (32)

Answer: A

This ECG indicates ECG evidence of severe hypothermia. The arrow on the ECG identifies the classic Osborn wave. With body temperatures less than 32°C (89.6°F), cardiovascular manifestations of hypothermia can be observed, including both systolic and diastolic dysfunction, dysrhythmias, hypotension, and shock. The correct intervention is rewarming. This ECG does not indicate any evidence of an acute coronary process with myocardial infarction, so thrombolysis is the incorrect selection. There is no arrythmia. The patient's ECG abnormalities are not manifestations of a toxic ingestion, so activated charcoal and sodium bicarbonate are also incorrect answers. After rewarming of this patient from 29°C (84.2°F), when the initial ECG was performed, the ECG changes improved. Figures 1-3 (below) show serial ECGs during rewarming to 32°C (89.6°F) and 38°C (100.°F), respectively, in a patient with hypothermia.

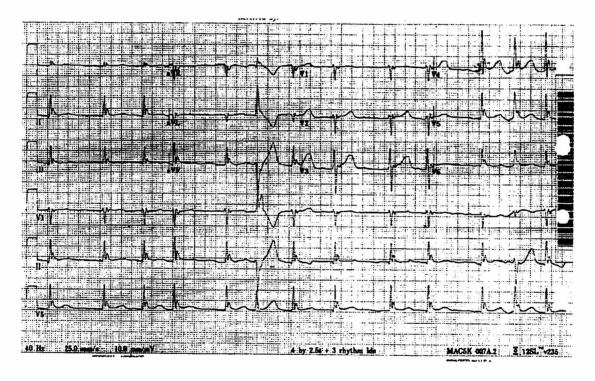


Figure 1. 29°C (84.2°F)

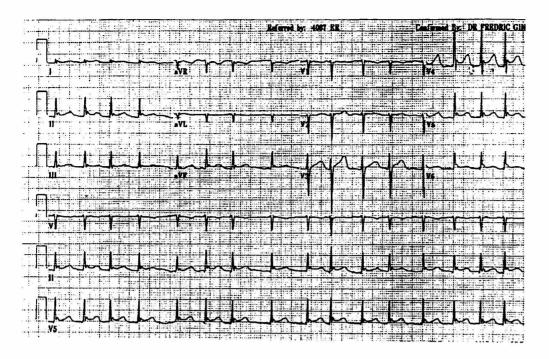


Figure 2. 32°C (89.6°F)

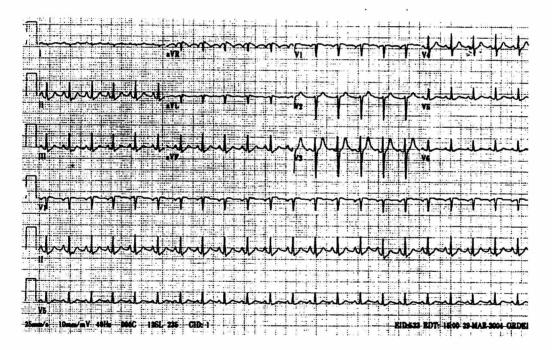


Figure 3. 38°C (100.4°F)

## **REFERENCES (32)**

Aslam AF, Aslam AK, Vasavada BC, Khan IA. Hypothermia: Evaluation, electrocardiographic manifestations, and management. *Am J Med.* 2006; 119:297-301.

Epstein E, Anna K. Accidental hypothermia. BJM. 2006; 332:706-709.

John AD, Fleisher LA. Electrocardiography: The ECG. Anesthesiol Clin. 2006; 24:697-715.

Jurkovich GJ. Environmental cold-induced injury. Surg Clin North Am. 2007; 87:247-267.

Wartofsky L. Myxedema coma. Endocrinol Metab Clin North Am. 2006; 24:50-51.

# SECTION 16: General Critical Care

## SECTION 16: GENERAL CRITICAL CARE

Instructions: For each question, select the most correct answer.

A 27-year-old female is brought to the emergency department after being found obtunded. She
was observed to be walking on a street and suddenly collapsed. A severe subarachnoid
hemorrhage is diagnosed by CT scan. She is admitted to the ICU. Upon examination, you find
that she has no brainstem reflexes; you suspect that she meets brain death criteria. You confirm
this later that day with an apnea test.

The patient's family is aware of the gravity of the prognosis and is in transit to your hospital. You are called to the bedside because the patient is markedly hypotensive. Physical examination reveals cool extremities and the systolic BP is in the 70s mm Hg.

Which one of the following should you do next?

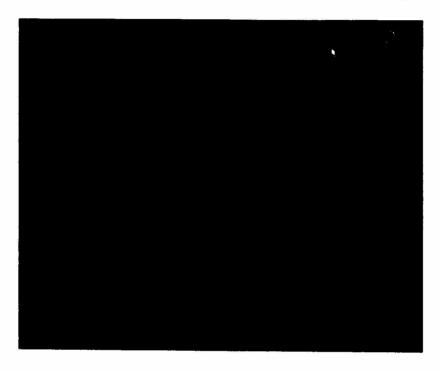
- A. Pronounce the patient dead and discontinue mechanical ventilation
- B. Write an order for no escalation of current therapy and withhold resuscitative efforts when cardiac arrests ensues
- C. Call for a consult from a physician from the neurosciences (neurology or neurosurgery) in order to corroborate your clinical impression of brain death
- D. Consider organ donation and insert a pulmonary artery catheter, titrating the patient's cardiovascular support to indices of cardiac filling pressure, cardiac index, and mean arterial pressure while awaiting the family's arrival
- 2. A patient needs central venous access. You choose the internal jugular vein as the site for line insertion. You plan to use ultrasound guidance in order to facilitate central venous catheter insertion and perform sonography to help locate the jugular vein in relationship to other landmarks and ensure patency prior to starting the procedure.

Which of the following is the most reliable discriminator for differentiating an arterial from a venous structure?

- A. Size
- B. Depth
- C. Compressibility
- D. Spatial relationship (i.e., medial vs lateral)
- E. Pulsatile movement

3. A patient with severe acute pancreatitis develops symptomatic hypotension and is treated with 2 L of crystalloid rapidly infused via peripheral IV lines. After the initial crystalloid resuscitation, the arterial pressure rises to 88/40 mm Hg and the patient remains oliguric. The patient's chest radiograph prior to intubation appears below.

You plan to insert a central venous catheter and measure the patient's central venous pressure.



Which of the following statements is most correct?

- A. A central venous pressure of 10 mm Hg indicates that IV fluids should be stopped
- B. A past medical history of congestive heart failure would indicate that IV fluids should be stopped
- C. The appearance of the lung fields on the chest radiograph indicates that IV fluids should be stopped
- D. The high volume of crystalloid already infused in a short period of time indicates that IV fluids should be stopped
- E. IV fluid resuscitation should be continued

- 4. Which one of the following statements about transfusion-related acute lung injury (TRALI) is most correct?
  - A. TRALI is the leading cause of transfusion-associated mortality
  - B. No specific patient-centered risk factors for TRALI have been identified
  - C. Risk of TRALI is independent of blood volume or rate of infusion
  - D. Establishing a temporal relationship between transfusion and lung injury is unnecessary for making the diagnosis
- 5. A 22-year-old man (70 kg) is burned in a house fire and brought to the emergency department. He suffered 2nd and 3rd degree burns over his back, one arm and one leg. Both extremity burns are circumferential. Which of the following crystalloid volumes, infused over the first 8 hours of resuscitation, is most correct?
  - A. 4 L
  - B. 6 L
  - C. 8 L
  - D. 12 L
- 6. Which one of the following is considered a core principle of medical ethics?
  - A. Compassion
  - B. Autonomy
  - C. Protection
  - D. Disclosure

- 7. You are asked to review the results of a clinical trial reporting the effects of a novel therapy in reducing mortality in patients with septic shock. Mortality in the placebo group of the trial was 60%, as compared with a mortality of 30% in the treatment group. How many patients will you need to treat with this new therapy in order to prevent 1 death from septic shock?
  - A. 2
  - B. 20
  - C. 3
  - D. 30
  - E. This number cannot be calculated without knowing the p value
- 8. A 59-year-old insulin-dependent diabetic is admitted to the ICU with a severe sepsis of unclear etiology. She is initially hypotensive, but BP has normalized with volume resuscitation alone. Because of the abdominal physical examination findings, a CT scan of the abdomen is obtained (see figure below).



Based on the CT scan and the clinical information presented, which one of the following is most correct?

- A. A percutaneous drainage procedure is indicated
- B. Surgical intervention is indicated for a ruptured diverticulum and associated abscess
- C. Nephrectomy is indicated
- D. Abnormal clotting function or trauma is the likely factor for the abnormal findings
- E. Emergent repair of the abdominal aortic aneurysm is indicated

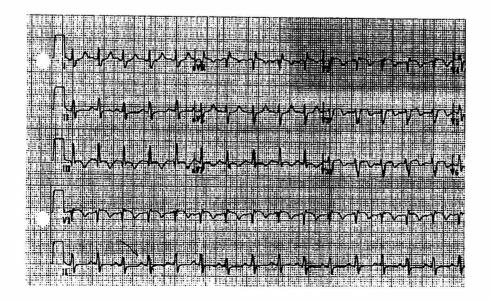
9. A 48-year-old female admitted to the hospital with a small bowel obstruction develops fever and hypotension on the medical ward. The patient had been non per os for the last 10 days, receiving parenteral nutrition through a right subclavian line. Vital signs show a temperature of 39.4°C (103.0°F), BP of 70/50 mm Hg, HR 115/min, RR 22/min, and SaO<sub>2</sub> 98% on 2 L nasal canula.

Examination reveals normal lungs, tachycardia, benign abdomen, and erythema with discharge around the site of the central venous catheter. The patient is started on empiric antibiotics and aggressive IV hydration. The central line is removed and a new line placed.

The patient is expeditiously transferred to the ICU. On arrival in the ICU, the patient's BP is 80/50 mm Hg with a HR of 110/min after receiving 1 L of normal saline solution IV. The patient is started on dopamine at 4  $\mu$ g/kg/min and continues to receive IV fluids. One hour after arriving to the ICU, the patient's BP is 110/60 mm Hg with a HR of 90/min. The patient continues to receive IV fluids but is not requiring dopamine.

Which one of the following answers is most correct in regards to the management of this patient?

- A. The patient should be treated with IV hydrocortisone 200 mg/d as a continuous infusion for 7 days
- B. The patient should receive methylprednisone 30 mg/kg IV q 6 hours for 2 days
- C. The patient should receive hydrocortisone 50 mg IV q 6 hours plus fludrocortisone 50 µg po qd
- D. This patient should not receive corticosteroids
- 10. A 46-year-old male with a past medical history significant for hypertension presents to the emergency department with complaints of acute shortness of breath and chest pain. Vital signs are significant for tachycardia and hypotension. An ECG is performed (see figure below).



Which of the following ECG findings is most likely to be present in this patient?

- A. Pericardial fluid with tamponade physiology
- B. Severe left ventricular systolic dysfunction
- C. Right ventricular dilation
- D. Regional motion abnormality in the anterior wall of the left ventricle
- E. Severe mitral regurgitation
- 11. A 75-year-old female is sent to the emergency department from a nursing home for evaluation of fever. Vital signs are temperature 39.0°C (102.2°F), HR 110/min, BP 128/82 mm Hg, RR 22/min. On examination, the patient is mildly confused; there are no focal deficits on neurologic examination and no neck rigidity. Lungs are clear to auscultation, heart sounds are regular, and abdominal examination is benign.

Initial laboratory data includes white blood cell count  $14,000/\mu$ L, hemoglobin 10, platelets 160,000, sodium 145 mEq/L, potassium 4.5 mEq/L, blood urea mitrogen 38 mg/dL, creatinine 1.9 mg/dL, and lactate 5.7 mmol/L.

Which of the following statements is most correct regarding this case?

- A. The elevated lactate is diagnostic of sepsis
- B. The elevated lactate is not important in the presence of a normal BP
- C. The lactate level is artificially elevated as a result of intravascular volume depletion
- D. The lactate level suggests sepsis-induced tissue hypoperfusion

12. You are treating a 56-year-old female admitted to the ICU after an out-of-hospital ventricular fibrillation cardiac arrest. The patient is on mechanical ventilation and receiving sedation and neuromuscular blocking agents while undergoing a therapeutic hypothermia protocol started 6 hours earlier.

Vital signs are temperature 33°C (91.4°F), HR 80/min, BP 110/70 mm Hg, RR 14/min. Over the last 2 hours, her urine output has increased from 150 mL/h to 800 mL/h. Laboratory data on admission to ICU (prior to hypothermia) sodium 142 mEq/L, potassium 4.5 mEq/L, blood urea nitrogen 18, creatinine 1.0 mg/dL. Current laboratory data are sodium 152 mEq/L, potassium 3.8 mEq/L, blood urea nitrogen 28, creatinine 1.2 mg/dL.

Which of the following statements is most correct regarding the treatment of this patient?

- A. The sodium changes are an artifact of hypothermia
- B. Desmopressin should be administered
- C. Fresh frozen plasma should be administered
- D. Fluids 0.9% normal saline solution should be administered to replace urinary losses
- E. The patient should be warmed to 37°C (98.6°F) over the next 6 hours
- 13. Which one of the following is true regarding the transfer of a critically ill patient to another facility?
  - A. The decision to transfer is the responsibility of the accepting physician at the receiving hospital
  - B. Minimum monitoring during transport should include continuous electrocardiography, intermittent pulse oximetry, intermittent BP and intermittent RR
  - C. Informed consent for transfer does not need to be obtained from the family or legal representative
  - D. The mode of transportation is determined by the accepting physician and hospital
  - E. A minimum of two people are required during transport in addition to the vehicle operators

14. A 28-year-old male was recently diagnosed with AIDS and pneumocystis pneumonia. He was started on trimethoprim-sulfamethoxazole and antiretroviral therapy as an outpatient. He presents to the hospital 2 weeks later with fever, myalgias, and rash. The rash is erythematous confluent macules covering a large extent of the body with blisters with sloughing in some areas. He is noted to have oral and conjunctival lesions.

Which one of the following statements is true regarding this patient's skin condition?

- A. The most common cause of death is infection
- B. Systemic corticosteroids should be administered as soon as possible
- C. The condition is most likely due to infection
- D. IV immunoglobulin has been shown to improve outcome
- E. Systemic antibiotics should be administered
- 15. A 45-year-old male with HIV underwent a flexible bronchoscopy for persistent right lower lobe infiltrate. His oropharynx was sprayed with a topical anesthetic, and he was given IV sedation with fentanyl 100 mcg and midazolam 2 mg. At the end of the 20 minute procedure, the patient was noted to be tachypneic and cyanotic. His HR was 90/min with RR of 22/min, BP 150/90 mm Hg, and SpO<sub>2</sub> 85%. Administration of 100% oxygen by nonrebreather mask did not affect his RR or cyanosis. The patient was transported to the ICU and initial arterial blood gas was pH 7.30, Paco<sub>2</sub> 32 mm Hg, and Pao<sub>3</sub> 360 mm Hg.

Which one of the following should be the next intervention?

- A. Obtain a spiral CT scan of chest
- B. Obtain a chest radiograph
- C. Obtain an arterial blood gas cooximetry
- D. Intubate the patient
- E. Administer flumazenil

- 16. Which of the following vitamins is needed to prevent the development of lactic acidosis due to parenteral nutrition in critically ill patients?
  - A. Vitamin B1 (thiamine)
  - B. Vitamin B6 (pyridoxine)
  - C. Vitamin K (phytonadione)
  - D. Vitamin A (retinol)
  - E. Vitamin C (ascorbic acid)
- 17. A 46-year-old female (60 kg) with acute pancreatitis is intubated for respiratory distress and sepsis. She is sedated with propofol (360 mg/h) in order to tolerate mechanical ventilation. Hypotension has been controlled with an infusion of norepinephrine. On ICU day 4, her arterial blood gas is 7.21/48/85 on Fio<sub>2</sub>0.5, pressure control 18, rate 12, positive end expiratory pressure 10 cm H<sub>2</sub>O. She has an increasing pressor requirement, her urine is heme-positive without red blood cells, and she is oliguric. What is the likely cause of her worsening clinical picture?
  - A. Sepsis
  - B. Acute respiratory distress syndrome
  - C. Adrenal insufficiency
  - D. Propofol infusion syndrome
  - E. Neuroleptic malignant syndrome
- 18. A 35-year-old woman is admitted to the ICU with fever and hypotension. Her only medical history is a 2-month history of fatigue and weight loss with no known etiology. On the day of admission, she had a syncopal episode while getting out of bed. On admission, her BP is 70/40 mm Hg, HR 110/min, temperature 39.2°C (102.5°F), and RR 18/min. Her physical examination is remarkable only for a thin woman in mild distress. Chest, cardiac, and abdominal examinations are unrevealing for any potential source of infection. Laboratory studies revealed sodium 128 mmol/L, potassium 5.6 mmol/L, chloride 102 mmol/L, HCO<sub>3</sub> 16 mmol/L, blood urea nitrogen 28 mg/dL, creatinine 1.5 mg/dL, and glucose 60 mg/dL. The white blood cell count and differential is normal. After 3 L of normal saline solution over 2 hours, the patient is alert but her BP is 80/50 mm Hg. Dopamine is initiated and titrated to 10 μg/kg/min to maintain BP 90/56 mm Hg. Empiric broad-spectrum intravenous antibiotics are started.

Which one of the following options is most appropriate at this time?

- A. Norepinephrine infusion and a decrease in the dopamine infusion
- B. Administer dexamethasone 4 mg IV
- C. Infuse 1 L of hetastarch
- D. Administer thyroxine IV and hydrocortisone 100 mg IV
- 19. A 44-year-old man is brought to the emergency department with lobar pneumonia and severe hypoxemia requiring endotracheal intubation and mechanical ventilation. He is sedated with etomidate and given succinylcholine for neuromuscular blockade to facilitate intubation. After successful intubation, he is sedated with a continuous infusion of lorazepam and given a long-acting neuromuscular blocker (vecuronium) due to dyssynchrony and high airway pressures.

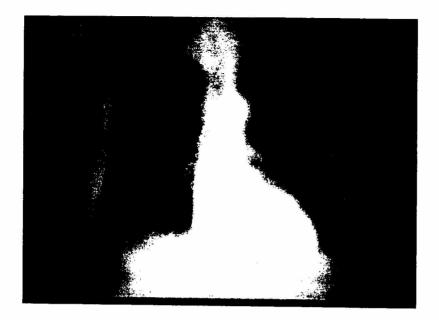
After transfer to the ICU, the arterial pressure (which had initially been mildly low and did not rise with intravascular volume expansion) drops sharply, requiring the initiation of vasopressors. His arterial pressure remains low (mean arterial pressure 50 mm Hg) despite a moderate dose of norepinephrine. Sepsis-associated relative adrenal insufficiency is suspected, and he is administered low-dose steroids, to which the BP responds, permitting a substantial decrease in the norepineprhine dose. Which of the following medications most likely has contributed to the hypotension in this patient?

- A. Etomidate
- B. Succinylcholine
- C. Lorazepam
- D. Vecuronium
- 20. A 65-year-old man with known lung carcinoma is found unresponsive. Clinically, he is euvolemic, and he weighs 60 kg. The serum sodium concentration is 108 mEq/L, the serum potassium concentration is 3.9 mEq/L, serum osmolality is 220 mOsm/kg H<sub>2</sub>O, the serum urea nitrogen concentration is 5 mg/dL, the serum creatinine concentration is 0.5 mg/dL, and urine osmolality is 600 mOsm/kg H<sub>2</sub>O.

Which one of the following is the most appropriate treatment at this time?

- A. Normal saline solution
- B. Demeclocycline, 400 mg, orally every 8 hours
- C. 3% Sodium chloride solution and free-water restriction
- D. Free-water restriction only

21. A 65-year-old male has right-side chest and upper abdominal pain. His supine chest radiograph is shown below.



Of the following choices, what test or procedure would be most useful at this time?

- A. Right-sided ECG
- B. Left lateral decubitus chest radiograph
- C. CT scan of mediastinum
- D. Insertion of nasogastric tube

22. A 58-year-old male is doing yard work on a very hot and humid day. He feels lightheaded and nauseous. He subsequently has multiple episodes of emesis. He feels better and lays down. He now notes chest discomfort that continues to worsen over the next 8 hours. He presents to the emergency department. Vital signs reveal mild hypertension, tachycardia, and tachypnea, with a temperature of 38.3°C (101.0°F). His white blood cell count is elevated, and his ECG is within normal limits. Chest radiograph shows a previously undiagnosed left-sided pleural effusion. A thoracentesis is performed.

Which one of the following abnormal fluid analyses is most likely to be present?

- A. Elevated glucose
- B. Presence of cholesterol crystals
- C. Elevated amylase
- D. Eosinophilia

- 23. Which one of the following statements is least correct concerning electrical injuries?
  - A. Delayed thrombosis of the peripheral arteries may convert a previous partial thickness injury into a full thickness injury
  - B. Direct renal injury is a common occurrence with truncal voltage injuries
  - C. Paralytic ileus and stress ulceration of gastric mucosa are the most common gastrointestinal complications of electrical injuries
  - D. A history of loss of consciousness in the field warrants inpatient admissions for ECG monitoring
- 24. To avoid malfunction of ventilators, which one of the following is the most appropriate minimum distance at which cellular phones should be used from intensive care ventilators?
  - A. Equal to or greater than 3 feet
  - B. Equal to or greater than 10 feet
  - C. Equal to or greater 20 feet
  - D. Outside of the ICU
  - E. Used nowhere in the hospital

25. A 59-year-old man is brought to the emergency department with sudden severe chest pain and dyspnea. Oxygen saturation is 90% by pulse oximeter (with high-flow supplemental oxygen delivered by face mask). RR is 35/min, with a HR 125/min. A CT scan (see figure below) was obtained.



Which one of the following diagnoses can be made from the CT scan?

- A. Pericardial effusion
- B. Pulmonary embolism
- C. Dissection of the ascending thoracic aorta
- D. Dissection of the descending thoracic aorta

#### **SECTION 16: GENERAL CRITICAL CARE**

#### **ANSWERS:**

1-D; 2-C; 3-E; 4-A; 5-B; 6-B; 7-C; 8-C; 9-D; 10-C; 11-D; 12-D; 13-E; 14-A; 15-C; 16-A; 17-D; 18-B; 19-A; 20-C; 21-B; 22-C; 23-B; 24-A; 25-B

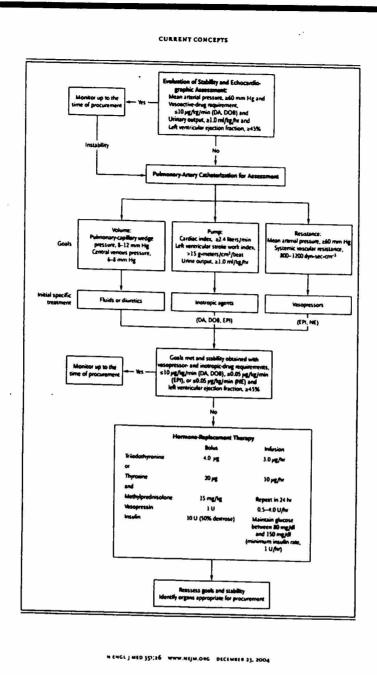
RATIONALE (1)

Answer: D

Optimal medical management of the potential organ donor is of paramount importance, as there continues to be a great disparity between the number of potential organ donors and that of actual donors. This was a potential organ donor who suffered an isolated neurologic injury. However, with profound hypotension and cardiovascular collapse in the time prior to organ procurement, the likelihood of successful outcomes for the recipients is threatened. Therefore, a strategy of optimal care of the potential organ donor is necessary, even after brain death has occurred.

Cardiovascular instability is a common sequela of death by neurologic criteria, as severe neurologic injury has been associated with deactivation of the sympathetic nervous system, with profound vasodilation and appropriately low levels of serum catecholamines. Vasodilation and cardiac dysfunction are common in hemodynamic instability following brain death.

The goals of managing the hemodynamically unstable potential organ donor are to achieve an optimization of the cardiac filling pressure, mean arterial pressure, and cardiac index in order to help ensure adequate tissue perfusion while using as low an amount as possible of vasoactive agents. A pulmonary catheter should be inserted, as optimization of hemodynamic status is not possible without these data. An expedited family meeting should be held in order to determine if the patient would have wanted to be an organ donor. If the family supports this decision, then the patient should be aggressively supported until organ procurement can be accomplished. If the family declines organ donation, then the patient should be taken off the ventilator immediately, as she has already met death by neurologic criteria. See Figure 1.



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Figure 1. An algorithm for support of the potential organ donor. Adapted from Wood and colleagues.

#### **REFERENCE (1)**

Beaulieu Y, Marik PE. Bedside ultrasonography in the ICU: Part 2. Chest 2005;128:1766-1781. Kusminsky RE. Complications of central venous catheterization. J Am Coll Surg 2007;204:681-696. Taylor RW, Palagiri AV. Central venous catheterization. Crit Care Med 2007;35:1390-1396. Wood KE, Becker BN, McCartney JG, et al. Care of the potential organ donor. N Engl J Med 2004;351:2730-2739.

RATIONALE (2)

Answer: C

Ultrasound guidance is a technique that can greatly facilitate the placement of central venous catheters. In literature, sonography has been shown to be superior to conventional landmarks (without sonography) in terms of success rates for first attempts at cannulation. Not only can ultrasound help to locate vessels, but it can also determine patency and identify whether or not there are any anatomical abnormalities specific to a given patient. For patients that have had multiple vascular procedures including central venous catheter placements in the past, ultrasound can be especially useful for identifying patency.

A compressible vein would indicate patency. A noncompressible vein may be thrombosed, and attempts at central venous catheterization may be difficult or even impossible. In order to differentiate an artery from a vein, compressibility is also a good discriminator.

Because of potential anatomic differences in individual patients, the spatial relationship is not necessarily reliable, nor is the size of the vessel. In order to help visualize the internal jugular vein, asking the awake patient to perform the valsalva maneuver will cause the vein to distend and may facilitate identification of the optimal location for needle placement or help identify that the vessel is indeed patent. See Figure 1.



Figure 1. Sonography in internal jugular vein cannulation. The larger (on the right) of the two vascular structures is the internal jugular vein and should be easily compressible. Reproduced from Self-Assessment in Multidisciplinary Critical Care: A Comprehensive Review. 5th ed. Chicago, IL: Society of Critical Care Medicine, 2003.

#### REFERENCE (2)

Stahmer SA and Mackowiak L. Ultrasound-guided procedures. In Roberts JR, Hedges J, eds., Clinical Procedures in Emergency Medicine. Philadelphia, PA: W.B. Saunders; 2005. RATIONALE (3)

Answer: E

This patient with severe acute pancreatitis has developed evidence of acute tissue hypoperfusion (hypotension and oliguria). At this time, the etiology of the circulatory insufficiency is not clear. Hypovolemia (due to either capillary leak or perhaps a hemorrhagic process in the pancreas) is possible as a source of the hypotension. Sepsis is also a potential concern. With signs of circulatory insufficiency, the patient needs aggressive resuscitation. After 2 L of crystalloid, the patient is still hypotensive and oliguric.

Although fluid resuscitation in a patient with a history of congestive heart failure will warrant some degree of caution, this patient has signs of circulatory insufficiency. Therefore, a restrictive fluid resuscitation strategy would not be prudent. Although the appearance of the lung fields on the chest radiograph suggests a pulmonary edema, a noncardiac pulmonary edema is the most likely etiology.

Recent administration of a high volume of crystalloid does not obviate the need for additional fluid resuscitation. Although a markedly low central venous pressure would indicate the patient needs intravascular volume expansion, a central venous pressure in this range (10 mm Hg) does not exclude the possibility that the patient's cardiac output is still preload-dependent.

Fluid resuscitation should be continued in this patient to resolve the signs of circulatory insufficiency. In a patient such as this one, adequate fluid resuscitation may result in the need for mechanical ventilation and positive end-expiratory pressure to maintain arterial oxygen saturation.

#### **REFERENCE (3)**

Magder S. Central venous pressure: A useful but not so simple measurement. *Crit Care Med* 2006:34:2224-2227.

Vincent JL, Weil MH. Fluid challenge revisited. Crit Care Med 2006;34:1333-1337.

RATIONALE (4)

Answer: A

Transfusion-related acute lung injury (TRALI) is the leading cause of blood transfusion-associated mortality. The risk of TRALI is dependent on both the volume of blood transfused and the rate of transfusion, with high volumes and high rates of infusion being more likely to precipitate this syndrome. As lung injury would only be attributed to TRALI in the presence of a temporal relationship (by convention ≤6 hours from the transfusion), a temporal relationship needs to be established in order to make the diagnosis.

Patient-centered risk factors for TRALI have been identified and include sepsis, shock, aspiration of gastric contents, disseminated intravascular coagulation, pneumonia, and burns.

#### **REFERENCE (4)**

Moore SB. Transfusion-related acute lung injury (TRALI): Clinical presentation, treatment, and prognosis. *Crit Care Med* 2006;34(Suppl):114-117.

Toy P, Popovsky MA, Abraham E, et al. Transfusion-related acute lung injury: definition and review. *Crit Care Med* 2005;33:721-726.

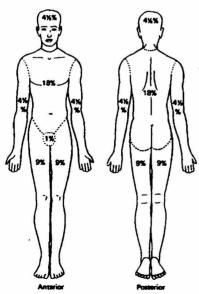
RATIONALE (5)

Answer: B

The patient has suffered 2nd and 3rd degree burns on 45% of his body surface area (BSA). The percent of the BSA that is involved in the burn can be estimated by the "rule of nines" (see figure below). This patient has circumferential (both anterior and posterior) burns to one arm and one leg, plus his entire back. Therefore, the percent BSA equals 9% + 18% + 18% = 45%.

The Parkland formula for calculating the volume of crystalloid resuscitation required over the first 24 hours is as follows: crystalloid volume = %BSA × weight (kg). This formula indicates that 12,600 mL of fluid will be required over the first 24 hours. Half of this fluid should be administered over the first 8 hours. This translates to 6 L of crystalloid over the first 8 hours of the patient's resuscitation. See the figure below.

A "more is better" approach to resuscitation is not necessarily prudent. Although patients with severe burns may require massive intravascular volume resuscitation, administering fluids in excess of what the patient actually requires may lead to complications. The most notable complications of over-resuscitating a burn patient would be compartment syndromes, either abdominal compartment syndrome, or perhaps, in this case, an extremity compartment syndrome. Given the circumferential burns that this patient has in his extremities, administering fluids in excess of what the patient needs will contribute to edema and could increase his risk of developing an extremity compartment syndrome.



#### **REFERENCE (5)**

Ipaktchi K, Arbabi S. Advances in burn critical care. *Crit Care Med* 2006;34(9 suppl):S239-S244 Klein MB, Hayden D, Elson C, et al. The association between fluid administration and outcome following major burn: A multicenter study. *Ann Surg* 2007;245:622-628.

RATIONALE (6)

Answer: B

The four core principles of medical ethics are beneficence, nonmaleficence, autonomy, and justice. Beneficence refers to the restoration of health, the preservation of life, and the relief of suffering. Nonmaleficence means to do no harm. Autonomy refers to respect for one's individuality, independence, and right to self-determination. Justice refers to the fair allocation of medical resources. These four principles can be used to analyze the complex bioethical issues that are common to ICU practice.

#### **REFERENCE (6)**

Carrese JA, Sugarman J. The inescapable relevance of bioethics for the practicing clinician. *Chest* 2006;130:1864-1872.

Luce JM. Ethical principles in critical care. JAMA 1990;263:696-700.

RATIONALE (7)

Answer: C

In order to answer this question, you need to calculate the number needed to treat (NNT). Results of clinical trials comparing an intervention to a placebo are reported in 1 of 3 ways: relative risk reduction (RRR), absolute risk reduction (ARR), or number needed to treat (NNT). In order to calculate these we need to know the controlled event rate (CER) and the experimental event rate (EER). The CER is the incidence of the study endpoint in the placebo group. The EER is the incidence of the study end point in the experiment or treatment group.

For our study, the CER is 60% and the EER is 30%. The results of trials are commonly reported as RRRs. RRR = (CER-EER)/CER, which is 50% for our study. ARR = CER – EER, which equals 30% in our study. NNT expresses the ARR in a user-friendlier format and is the inverse of the ARR (NNT = 1/ARR). In our case, NNT = 1/3/10 = approximately 3.

The p value measures statistical significance and is not required for calculating the number needed to treat. Although p values are commonly quoted when evaluating clinical trials, it is a statistical measurement of the likelihood that the results might have occurred by chance. A statistically significant p value does necessarily imply a significant clinical effect.

#### **REFERENCES (7)**

- Greenhalgh T. Statistics for the non-statistician II: significant relations and their pitfalls. Available at http://bmj.com/cgi/content/full/315/7105/422. Accessed June 13, 2007.
- Guyatt GH, Sinclair J, Cook DJ, et al. User's guide to the medical literature XVI: how to use a treatment recommendation. *JAMA* 1999;281:1875.
- McGovern D, Valori RM, Summerskill WSM, Levi M. Key Topics in Evidence-Based Medicine. Oxford, UK: BIOS Scientific Publishers Ltd; 2001

RATIONALE (8)

Answer: C

The CT scan demonstrates a retroperitoneal infection due to emphysematous pyelonephritis (EP) of the right kidney. In this patient with hemodynamic stability, immediate surgery is paramount. EP has the characteristic CT scan findings of air in the kidney (radiolucencies), usually with a characteristic subcapsular component. EP occurs most commonly in insulin-dependent diabetics, is usually due to Gram-negative organisms, and is associated with urosepsis. Diagnosis may also be suggested by ultrasound.

The CT scan fails to demonstrate any well-delineated abscess cavity that would require percutaneous drainage. Anecdotal reports of the success of percutaneous drainage of EP exist, but most sources recommend nephrectomy.

There is no evidence of an aortic aneurysm, nor is there evidence of a retroperitoneal hemorrhage.

#### **REFERENCES (8)**

- Cook DJ, Achong MR, Dobranowski J. Emphysematous pyelonephritis: complicated urinary tract infection in diabetes. *Diabetes Care*1989;12:229.
- Donovan MG, Logan H, Angus D. Emphysematous pyelonephritis: diagnosis by ultrasound. Br J Urol 1989;63:213.
- Hall JR, Choa RG, Wells IP. Percutaneous drainage in emphysematous pyelonephritis—an alternative to major surgery. *Clin Radiol* 1988;39:622.
- Roberts JA. Pyelonephritis, cortical abscess, and perinephric abscess. *Urol Clin North Am* 1986;13:637.

RATIONALE (9)

Answer: D

The use of corticosteroids in sepsis has received increased attention in the last several years. In the 1980s, high doses of corticosteroids for short periods of time were utilized with the intent of modulating the inflammatory response to sepsis. Randomized trials demonstrated that this therapeutic approach was not beneficial for patients with sepsis, and the use of high dose corticosteroids as anti-inflammatory mediators in sepsis was abandoned.

More recently, the concept of utilizing corticosteroids for their mineral corticoid effects and their ability to regulate basal vasomotor tone was explored. The concept of relative adrenal renal insufficiency and the potential benefit of replacing corticosteroids in patients with septic shock has been the subject of several clinical trials. Studies have consistently demonstrated that in patients who are in septic shock and on vasopressors, despite adequate fluid resuscitation, the treatment with doses of hydrocortisone equivalent to approximately to 200 mg/day given in 4 divided doses intravenously or as a continuous infusion will improve BP and decrease vasopressor requirements.

Annane and colleagues have demonstrated that a 7-day course of hydrocortisone plus fludrocortisone would improve mortality in patients who failed to respond to a cosyntropin stimulation test and are deemed to have relative adrenal insufficiency. Although there is still debate in the literature on the best method to identify patients with possible relative adrenal insufficiency, it is currently recommended that corticosteroids at a dose of 200 mg/day, given either as a continuous infusion or divided doses, be considered for patients who require vasopressors for septic shock despite adequate fluid resuscitation.

However, this patient should not receive corticosteroids. Although the patient has severe sepsis and sepsis-induced hypotension, her BP has improved with IV fluids. The patient was only on dopamine for a very short period of time. Patients who are studied in the reference trials were on vasopressors for more than 6 hours.

#### **REFERENCES (9)**

- Annane D, Sebille V, Charpentier C, et al. Effect of treatment with low doses of hydrocortisone and fludrocortisone on mortality in patients with septic shock. *JAMA* 2002;288:862-871.
- Annane D, Bellisaant E, Bollaert PE, et al. Corticosteroids for severe sepsis and septic shock: a systematic review and meta-analysis. *BMJ* 2004;329:480.
- Dellinger RP, Carlet JM, Masur H, et al. Surviving Sepsis Campaign Management Guidelines Committee: Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med* 2004;32:858-873.
- Minneci PC, Deans, KJ, Banks SM, et al. Meta-analysis: the effect of steroids on survival and shock during sepsis depends on the dose. *Ann Intern Med* 2004;141:47-56.
- Russell JA. Drug therapy. Management of sepsis. N Engl J Med 2006;355:1699-1713.

RATIONALE (10) Answer: C

The ECG shown above is significant for sinus tachycardia with an incomplete right bundle branch block and evidence of an S wave in lead 1, a Q wave in lead 3, and a inverted T wave in lead 3 (S1, Q1, T3). These ECG findings, in association with the above clinical presentation, are highly suggestive of a severe pulmonary embolism (PE). Findings that have been reported in patients with a PE on an ECG include dilation of the right pulmonary artery, dilation of the right ventricular diameter, and increased right ventricular end-systolic dimension-left ventricular end-diastolic ratio. Other studies have reported a detection of a thrombus within the right pulmonary artery with ECG.

Pericardial effusion and evidence of a cardiac tamponade would be expected in patients with an ECG suggestive of fluid around the heart, which is not seen in this patient.

Decreased left ventricular systolic dysfunction would be associated in the presentation typical of acutely decompensated heart failure, or in a patient who has significant hypertension. None of these clinical scenarios are evident in this case.

Regional wall motion abnormalities on the left ventricle are commonly seen with acute coronary ischemia which this patient does not have.

Finally, evidence of mitral regurgitation would be seen in patients with PE and acute abnormal papillary muscle dysfunction, none of which are present in this patient.

#### **REFERENCES (10)**

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- Buckley JD and Popovich J. Pulmonary embolism. In Dellinger P, Parrillo J, eds. *Crit Care Medicine*. 2nd ed. Philadelphia, PA: Mosby; 2001.
- Dalen JE. Pulmonary embolism: What have we learned since Virchow?: Natural history, pathophysiology, and diagnosis. *Chest* 2002; 122:1440-1456.
- Dalen JE. Pulmonary embolism: What have we learned since Virchow?: Treatment and prevention. *Chest* 2002; 122:1801.
- Fedullo PF, Tapson VF. The evaluation of suspected pulmonary embolism. *N Engl J Med* 2003;349:1247-1256.

RATIONALE (11)

Answer: D

Current guidelines for the management of severe sepsis and septic shock recommend that patients identified as having sepsis have their serum lactate checked as part of their initial work-up. Studies have demonstrated that an increased lactate in patients with severe sepsis is associated with increased mortality. In addition, an elevated lactate seems to identify patients with sepis-induced tissue hypoperfusion, even when their vital signs are considered to be normal. In a subgroup of patients from the Early Goal-Directed Therapy study by Rivers and colleagues, patients who had an elevated lactate in the presence of a mean arterial pressure >100 had severely impaired mixed venous central oxygen saturations and a very high mortality associated with sepsis.

It is important to recognize that an elevated lactate does not confirm a diagnosis of sepsis. Lactate in patients with sepsis should be used to risk-stratify patients, and can identify patients who require more aggressive hemodynamic support. It is very important to understand that even patients who have a normal BP with severe sepsis, but exhibit an increased lactate, may have significant tissue hypoperfusion. Of the given answers, answer D, the lactate level suggests sepsis-induced tissue hypoperfusion, is the most correct.

#### REFERENCES (11)

Dellinger RP, Carlet JM, Masur H, et al. Surviving Sepsis Campaign Management Guidelines Committee: Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med* 2004;32:858-873.

Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001;345:1368-1377.

Sepsis Resuscitation Bundles. Available at: http://www.ihi.org. Accessed June 13, 2007.

Shapiro NI, Howell MD, Talmor D, et al. Serum lactate as a predictor of mortality in emergency department patients with infection. *Ann Emerg Med* 2005;45:524-528.

RATIONALE (12)

Answer: D

Based on the results of two randomized clinical trials, the Advanced Life Support Task Force of the International Liaison Committee on Resuscitation (ILCOR) made recommendations regarding the implementation of therapeutic hypothermia after cardiac arrest. Current indications include unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest, when the initial rhythm was ventricular fibrillation (VF). These patients should be cooled to 32°C (89.6°F) to 34°C (93.2°F) for 12-24 hours. The ILCOR has also stated that such cooling may also be beneficial for other rhythms or in-hospital cardiac arrest.

With the increasing use of therapeutic hypothermia in patients who survive cardiac arrest, it is important to recognize some of the practical aspects and side effects of implementing this therapeutic option. In this question, the patient is an appropriate candidate for therapeutic hypothermia. After 2 hours of cooling, the patient demonstrates a significant increase in urine output and an increase in the serum sodium from 142 to 152 mEq/L.

The physiologic effects of hypothermia are diverse, and include metabolic, endocrine, cardiovascular, hematological and renal changes. As the temperature drops below 35°C (95°F), there is an increase in diuresis associated with tubular dysfunction and electrolyte losses. The diuresis evidenced by this patient and the increase in sodium are direct consequences of hypothermia. These are physiologic effects that should be anticipated and, therefore, replacing the urinary losses with normal saline solution is the correct response.

Although some neurological catastrophes can be associated with a syndrome of inadequate antidiuretic hormone secretions, there is no evidence of that phenomenon in this patient. Desmopressin is not indicated.

Patients who are cooled with therapeutic hypothermia will present hematological abnormalities that include a decreased platelet count with impaired platelet function and other coagulation abnormalities. However, this patient has no symptoms that would require the administration of fresh frozen plasma.

Finally, there is no reason to rewarm the patient at this point. The goal should be to complete the 12-24 hours of therapeutic hypothermia, and then rewarm the patient. Once again, it is important to recognize the physiologic effects as therapeutic hypothermia is implemented, so they can be anticipated and corrected as they occur, by very aggressively replacing lost fluids with normal saline or other isotonic fluids and by following the electrolytes very closely.

#### **REFERENCES (12)**

- Arrich J, The European Resuscitation Council Hypothermia After Cardiac Arrest Registry Study Group. Crit Care Med 2007;35:1041-1047.
- Polderman K. Application of therapeutic hypothermia in the intensive care unit. *Intensive Care Med* 2004;30:757-769.
- Williams Gr, Spencer FC. The clinical use of hypothermia following cardiac arrest. *Ann Surg* 1958;148:462-468.

RATIONALE (13)

Answer: E

Guidelines from the Society of Critical Care Medicine suggest a minimum of 2 people, other than the vehicle operators, are needed. The transporting team should be capable of providing airway management, IV therapy, arrhythmia recognition, and basic and advanced life support. Additional expertise may be required in unstable patients.

Minimum monitoring requirements during transport include continuous electrocardiography and pulse oximetry and periodic regular BP and RR. Additional monitoring such as intraarterial BP or central venous pressure measurements may be needed in some patients.

The decision to transfer a critically ill patient rests with the referring physician after careful consideration of the potential risks and benefits. The referring physician is also responsible for the resuscitation and stabilization of the patient to the best of their ability.

The mode of transportation is determined by the transferring physician based on the patient's condition, weather conditions, interventions needed for life support during the transfer, and the availability of personnel and modes of transport.

Informed consent should be obtained from the patient or an appropriate representative after a discussion of risks and benefits. Life-threatening circumstances may preclude such a discussion, but the record should document the reasons for the transfer and for not obtaining consent.

#### **REFERENCES (13)**

Australasian College for Emergency Medicine, Joint Faculty of Intensive Care Medicine, Australian and New Zealand College of Anaesthetists. Minimum standards for transport of critically ill patients. *Emerg Med* 2003;15:197–201.

Wallace PG, Ridley SA. ABC of intensive care: transport of critically ill patients. *BMJ* 1999;319:368-371.

Warren J, Fromm RE, Orr RA, et al. Guidelines for the inter- and intrahospital transport of critically ill patients. Crit Care Med 2004;32:256-262.

RATIONALE (14)

Answer: A

The clinical situation and the description of the mucocutaneous lesions make a diagnosis of Stevens-Johnson syndrome/toxic epidermal necrolysis (TEN) most likely in this patient. In addition, HIV-infected patients are predisposed to this condition, probably due to an increased exposure to drugs. The majority of cases (>80%) are attributed to drugs, with antibiotics (sulfonamides, quinolones, etc) and anticonvulsants (phenytoin, carbamazepine, etc) being common. Infection with viruses or Mycoplasma is much less common.

Stevens-Johnson and TEN are thought to be due to an immune response to antigenic complexes formed between drug metabolites and host tissue. Both are part of the same disease spectrum with TEN defined as having >30% epidermal loss. Immediate withdrawal of the suspected drug and supportive care are the primary interventions. Patients with significant skin involvement should be cared for in a burn or wound care unit or ICU. Care is needed to maintain fluid and electrolyte balance, normothermia, nutrition, and pain relief, as well as monitoring for evidence of infection. Ophthamologic evaluation should also be obtained.

Routine use of systemic antibiotics is not recommended in the absence of suspected infection. Infection with *Staphylococcus aureus* and *Pseudomonas aeruginosa* is the most common cause of death.

The general consensus is that corticosteroids should not be administered, although adequate clinical trials have not been performed. Likewise, IV immunoglobulin has not been evaluated in clinical trials for evidence of benefit. Case series of immunoglobulin use have reported mixed results.

#### **REFERENCES (14)**

Bachot N, Roujeau J-C. Intravenous immunoglobulins in the treatment of severe drug eruptions. Curr Opin Allergy Immunol 2003;3:269-274.

French LE, Trent JT, Kerdel FA. Use of intravenous immunoglobulin in toxic epidermal necrolysis and Stevens-Johnson syndrome: our current understanding. *Int Immunopharmacol* 2006;6:543-549.

Hussain W, Craven NM. Toxic epidermal necrolysis and Stevens-Johnson syndrome. *Clin Med* 2005;5:555-558.

RATIONALE (15)

Answer: C

This patient most likely has benzocaine-induced methemoglobinemia; co-oximetry would measure the methemoglobin level. Measured arterial oxygen saturation that is much lower than that calculated for the alveolar oxygen tension also is suspicious for methemoglobinemia. The methemoglobin is incapable of carrying oxygen, resulting in functional anemia and an impaired tissue oxygen delivery.

Methemoglobin is formed when the ferrous iron in the heme molecule is oxidized to the ferric state. The normal mechanisms that convert methemoglobin back to hemoglobin can be overwhelmed by many oxidant drugs (such as benzocaine, lidocaine, nitroglycerin, nitrites, antimalarials, aniline dyes, and dapsone), resulting in toxic methemoglobinemia.

The hallmark of methemoglobinemia is central cyanosis, which usually appears at methemoglobin levels above 15%. Patients are usually asymptomatic until the concentration exceeds 20 to 30%. Between 20 and 50%, they may experience weakness, malaise, nausea, vomiting, headache, dyspnea, and tachycardia. Once levels exceed 50%, patients may develop lethargy, dizziness and stupor. At these high levels, there is inadequate oxygenation of tissues, which may result in acidosis, circulatory failure, cardiac arrhythmias, seizures and coma. Levels above 70%, although rare, are associated with a high incidence of mortality.

The pulse oximeter cannot be used to accurately assess oxygen saturation in a patient with methemoglobinemia. At low concentrations of methemoglobin (<10%), oxygen saturation will be underestimated. At high concentrations, the pulse oximeter will read 85% saturation regardless of the relative amounts of oxyhemoglobin and deoxyhemoglobin present.

The diagnosis should be entertained when cyanosis, unresponsive to 100% oxygen therapy, appears suddenly, especially when exposure to an oxidant drug is established. Transesophageal echocardiography and bronchoscopy are the most common procedures reported to be associated with methemoglobinemia. Diagnosis is confirmed by multiple-wavelength co-oximetry.

Most cases require only decontamination and supportive care. Methylene blue is the specific antidote but should be reserved for more severe cases (usually when methemoglobin levels exceed 30% to 40%) or if comorbid conditions make mild hypoxia unadvisable. Methylene blue is contraindicated in patients with glucose-6-phosphate dehydrogenase deficiency due to potential hemolysis. Exchange transfusion or hemodialysis may be indicated in patients who fail to respond to methylene blue.

Intubation is usually not needed if the diagnosis of methemoglobinemia is determined and treatment iniated. A chest radiograph or CT scan would add little to the patient's care. Respiratory depression due to midazolam is not likely, due to the elevated RR, so flumazenil is not indicated.

#### **REFERENCES (15)**

- Coleman MD, Coleman NA. Drug-induced methemoglobinaemia. Treatment issues. *Drug Saf* 1996;14:394-405.
- Moore TJ, Walsh CS, Cohen MR. Reported adverse event cases of methemoglobinemia associated with benzocaine products. *Arch Intern Med* 2004;164:1192-1196.
- Sharma V, Haber A. Acquired methemoglobinemia: a case report of benzocaine-induced methemoglobinemia and a review of literature. Clin Pulm Med 2002;9:53-58.
- Wright WO, Lewander WJ, Woolf AD. Methemoglobinemia: etiology, pharmacology and management. *Ann Emerg Med* 1999;34:646-656.

RATIONALE (16) Answer: A

Trace elements and vitamins, also know as micronutrients, play an important role in various pathways and reactions that have an increased activity during critical illness. In addition, some critical conditions may be associated with increased losses of trace elements. It is very important to ensure that nutritional support for critically ill patients fulfills daily requirements for micronutrients. When the volume of enteral feeds is <1,500 mL for several days, or in severe conditions where there are increased requirements and large losses of trace elements, IV administration of vitamins B1, B6, C, E, and A may be beneficial.

Of the options given in the questions, vitamin B1 (100 mL/day) is essential to avoid lactic acidosis and other complications such as Beri Beri, Korsakoff syndrome and Wernicke encephalopathy. Patients with severe malnutrition, and patients with a history of chronic alcoholism, are at particular risk for developing these complications when glucose administration is initiated.

#### **REFERENCES (16)**

- ASPEN Board of Directors. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. *J Parenter Enteral Nutr* 1993;17:1SA-26SA.
- Bistrian BR, McCowen KC. Nutritional and metabolic support in the adult intensive care unit: Key controversies. Crit Care Med 2006;34:1525-1531.
- Burgor M, et al. Trace Elements and vitamins. In Pichard C, Kudsk K, eds. *Nutritional Support to Pharmacologic Nutrition in the ICU*. Updated in *Intensive Care Medicine and Emergency Medicine*. Verlag, London: Springer Verlag; 2000:66-79.
- Cerra FB, Benitez MR, Blackburn GL, et al. Applied nutrition in ICU patients. A consensus statement by the American College of Chest Physicians. *Chest* 1997;111:769-778

RATIONALE (17)

Answer: D

The propofol infusion syndrome (PRIS) was initially described for pediatric patients undergoing long-term sedation using propofol infusions. Subsequently, several reports of PRIS have been reported for adults. The syndrome is characterized by cardiac failure, rhabdomyolysis, severe metabolic acidosis, and renal failure. The etiology of PRIS is likely multifactorial. Catecholamines and glucocorticoids coupled with systemic inflammation have been considered "priming factors," with propofol itself as the "triggering" factor. The cellular mechanism may be the impairment of free fatty acid utilization that creates an imbalance in energy demand and utilization leading to cardiac and skeletal muscle necrosis.

It has been suggested that propofol should not be used in high doses (>5 mg/kg/h) for periods >48 hours to minimize the risk for PRIS. However, PRIS has been reported in a patient receiving steroids after the administration of 9 mg/kg/h for 3 hours followed by a postoperative infusion of 2.3 mg/kg/h.

#### **REFERENCES (17)**

Kang TM. Propofol infusion syndrome in critically ill patients. Ann Pharmacother 2002;36:1453-1456.
Liolios A, Guerit JM, Scholtes JL, et al. Propofol infusion syndrome associated with short-term large-dose infusion during surgical anesthesia in an adult. Anesth Analg 2005;100:1804-1806.
Vasile B, Rasulo F, Candiani, et al. The pathophysiology of propofol infusion syndrome: a simple name for a complex syndrome. Intensive Care Med 2003; 29:1417-1425.

RATIONALE (18)

Answer: B

The clinical findings in this patient are consistent with acute adrenal insufficiency. Clinical findings may include hypotension, orthostasis, fatigue, fever, and weight loss. In addition, patients may have nausea, vomiting, weight loss, and abdominal pain. Laboratory findings that support a diagnosis of adrenal insufficiency include hyponatremia, hyperkalemia, prerenal azotemia, hypoglycemia and normal anion gap acidosis. The most common causes of primary adrenal insufficiency are autoimmune destruction, infection, and metastatic disease. Other causes include bilateral adrenal hemorrhage, particularly in the critically ill patient with disseminated intravascular coagulation, infiltrative disorders, and some drugs (i.e., ketoconazole).

Secondary adrenal insufficiency due to failure of the pituitary to secrete adrenocorticotropic hormone (ACTH) is most commonly due to withdrawal of corticosteroids but can also result from tumors, head trauma, central nervous system radiation, and infiltrative diseases. Adrenal crisis may be confused with septic shock since the manifestations are similar. Clues to the presence of adrenal insufficiency include the lack of predisposing conditions for infection, the lack of an obvious source, and the poor response to intravenous fluids and vasopressor agents.

The treatment of adrenal crisis includes volume resuscitation with glucose-containing fluids and immediate administration of corticosteroids. The most appropriate management of this patient includes immediate administration of dexamethasone, which will not interfere with determination of plasma cortisol, and the performance of a short ACTH stimulation test.

Administration of colloids or a change in vasopressor agent is unlikely to have any significant effect in this patient. This patient's clinical presentation is not consistent with hypothyroidism so thyroxine is not indicated.

The short ACTH stimulation test involves the intravenous administration of cosyntropin (synthetic ACTH) 250  $\mu$ g with plasma cortisol determinations at 0, 30, and 60 minutes. A normal response requires a 30-minute or 60-minute level of 20  $\mu$ g/dL, and an increase of 7  $\mu$ g/dL above the basal level. Once the test is completed, hydrocortisone in doses of 300 mg/day, in divided doses, can be instituted.

#### **REFERENCES (18)**

- Bouachour GP, Tirot N, Varache JP, et al. Hemodynamic changes in acute adrenal insufficiency. *Intensive Care Med* 1994;20:138-141.
- Cooper MS, Stewart PM. Corticosteroid insufficiency in acute ill patients. N Engl J Med 2003;348:727-734.
- Parker N, Taylor RW. Adrenal insufficiency in the critically ill patient. In *Critical Care Medicine*, 2nd ed. Parrillo JE, Dellinger RP, eds. St. Louis, MO: Mosby; Inc., 2001, 1225-1234.
- Salvatori R. Adrenal insufficiency. JAMA 2005;294:2481-2488.
- Shenker Y, Skatrud JB. Adrenal insufficiency in critically ill patients. Am J Respir Crit Care Med 2001;163:1520-1523.
- Zaloga GP, Marik P. Hypothalamic-pituitary-adrenal insufficiency. Crit Care Clin 2001;17:25-41.

RATIONALE (19)

Answer: A

The patient has sepsis and is at risk for relative adrenal insufficiency that could be worsened or precipitated by a medication that effects the adrenal axis. Etomidate can inhibit adrenal steroidogenesis and would be the most likely agent in this scenario to precipitate or worsen adrenal dysfunction. When intubating a patient with severe sepsis or septic shock, it would be prudent to either (a) select an alternate sedative agent for induction, or (b) have a heightened awareness of the need to test the adrenal axis (or administer steroid replacement therapy) in the event that the patient develops refractory hypotension.

#### **REFERENCES (19)**

Jackson WL, Jr. Should we use etomidate as an induction agent for endotracheal intubation in patients with septic shock? a critical appraisal. *Chest* 2005;127:1031-1038.

Malerba G, Romano-Girard F, Cravoisy A, et al: Risk factors of relative adrenocortical deficiency in intensive care patients needing mechanical ventilation. *Intensive Care Med* 2005;31:388-92.

RATIONALE (20)

Answer: C

This patient has tumor-induced, inappropriate secretion of antidiuretic hormone. The diagnosis is based on the findings of hypotonic hyponatremia and concentrated urine in a euvolemic patient, the absence of a history of diuretic use, and the absence of a clinical picture consistent with severe hypothyroidism or hypoadrenalism. Infusion of hypertonic saline solution should be considered for patients who have severe hyponatremia with neurologic symptoms, concentrated urine (osmolality, less than or equal to 200 mOsm/kg H<sub>2</sub>O), and clinical euvolemia or hypervolemia. Therefore, the treatment plan for this patient would include water restriction and infusion of 3% sodium chloride. Because syndrome of inappropriate secretion of antidiuretic hormone can be thought of as a state of inappropriate fixed concentration of the urine, it follows that administration of loop diuretics will aid in management by inhibiting tubular concentrating mechanisms in the ascending limb of the loop of henle. Also, their administration may ameliorate the gain in total body sodium that comes with the administration of hypertonic saline. However, loop diuretics are usually reserved for patients who are clinically hypervolemic.

The expected change in serum sodium by the addition of 1 L of infusate can be calculated by using the following formula:

Change in serum Na = (infusate Na - serum Na) / (total body water + 1) (Where total body water (TBW) =  $0.6 \times \text{meight}$  (kg) for males;  $0.5 \times \text{meight}$  (kg) for females) According to formula, the retention of 1 L of 3% sodium chloride, which contains 513 mEq of sodium, will increase serum sodium concentration by 10.9 mEq/L ( $[513-108] \div [36+1]=10.9$ ). The initial goal is to increase the serum sodium concentration by 5 mEq/L over the next 12 hours. Therefore, 0.46 L of 3% sodium chloride ( $5 \div 10.9$ ), or 38 mL per hour, is required. These formulas should be used with caution, however, because patients are not static systems (because of ongoing losses, etc). Frequent monitoring of electrolytes and appropriate adjustments of therapy are necessary.

Demeclocycline induces a state of nephrogenic diabetes insipidus and increased free-water loss, gradually returning serum osmolarity toward normal. It is not appropriate for acute management of hyponatremia and is best used in patients with chronic syndrome of inappropriate secretion of antidiuretic hormone who cannot tolerate strict free-water restriction.

#### **REFERENCE (20)**

Adrogué HJ, Madias NE. Primary care: hyponatremia. N Engl J Med 2000; 342:1581-1589.

RATIONALE (21)

Answer: B

This chest radiograph demonstrates a deep sulcus sign in the right costophrenic angle. This is one of the signs of a pneumothorax on supine chest radiographs. Supine and semi-recombinant films have poor sensitivity for detecting pneumothoraces. We are trained to recognize what is called a visceral pleural line as an indicator of pneumothorax. The visceral pleural line is the thin white line (the visceral pleura) that with a pneumothorax is pulled away from the chest wall, with lung parenchyma (radiolucency with small lines) on the inside, and air (radiolucency without small white lines) on the outside.

The lack of sensitivity of supine films is particularly problematic, as there is a high progression to tension pneumothorax, likely because of the association of supine chest radiographs with mechanical ventilation. There are, however, some subtle radiographic clues to the presence of pneumothorax on supine chest radiographs. A deep lateral costophrenic angle may be the only sign of pneumothorax in over 50% of supine position radiographs. This is because the most anterior portion of the pleura is at the base. On occasions, the only evidence of a pneumothorax is a generalized hyperlucency over the base of the lung.

When the question of pneumothorax is raised, either due to deep sulcus sign or basilar hyperlucency, there are two options. You can use a lateral decubitus film, with the lung of concern up, to note presence or absence of air outside the pleural space in the nondependent area of the lung. Or you can sit the patient as upright as possible for another film. In addition to basilar hyperlucency and deep

sulcus sign, supine radiographic findings of pneumothorax include the depression of the diaphragm, a sharp mediastinal or diaphragmatic contour, a double diaphragm sign, a distinct cardiac apex, and apical pericardial fat tags (lobulated/rounded).

#### **REFERENCES (21)**

Baumann MH. Management of spontaneous pneumothorax. Clin Chest Med 2006;27:369-381. Qureshi NR, Gleeson FV. Imaging of pleural disease. Clin Chest Med 2006;27:193-213.

RATIONALE (22)

Answer: C

This patient has Boerhaave syndrome. Boerhaave syndrome, or spontaneous esophageal rupture, is an uncommon condition associated with a sudden increase in intra-esophageal pressure. In this case, it was the vomiting that triggered the Boerhaave syndrome.

Esophageal perforations usually involve the left thoracic cavity, as anatomically the distal esophagus is directly beneath the left thoracic pleura. Plain radiography of the chest may reveal hydrothorax, hydropneumothorax, or mediastinal emphysema.

Pleural effusion is characterized by an increased amylase of salivary etiology. Contrast study is required to locate the exact site of the perforation. Water soluble contrast is used initially; if that study is inconclusive, it is repeated with barium. Surgery is typically required. Successful surgeries have included primary closure, reinforced primary closure, and mediastinal drainage alone.

#### **REFERENCES (22)**

- Adams BD, Sebastian BM, Carter J. Honoring the Admiral: Boerhaave-van Wassenaer's syndrome. Dis Esophagus 2006;19:146-151.
- Hill AG, Tiu AT, Martin IG. Boerhaave's syndrome: 10 years experience and review of the literature. ANZ J Surg 2003;73:1008-1010.
- Khan AZ, Strauss D, Mason RC. Boerhaave's syndrome: diagnosis and surgical management. Surgeon 2007;5:39-44.
- Ng CS, Mui WL, UYim AP. Barogenic esophageal rupture: Boerhaave syndrome. Can J Surg 2006;49:438-439.
- Ringstrom E, Freedman J. Approach to undifferentiated chest pain in the emergency department: a review of recent medical literature and published practice guidelines. *Mt Sinai J Med* 2006;73:499-505.

RATIONALE (23)

Answer: B

Vascular injuries from electrical current can present either immediately or as a delayed complication. Medical wall damage can result in delayed thrombosis. Direct renal injury rarely occurs during electrical injuries. Acute renal failure, however, may result secondary to rhabdomyolysis. Gastrointestinal injuries are rare. Paralytic ileus and stress ulceration of gastric mucosa are the most frequently seen gastrointestinal complications. Admissions for monitoring are required if any of the following is present: (1) history of loss of consciousness or cardiac arrest, (2) cardiac arrhythmia in the field or in the emergency department, and (3) abnormal ECG.

#### **REFERENCES (23)**

Arnoldo B, Klein M, Gibran NS. Practice guidelines for the management of electrical injuries. J Burn Care Res 2006;27:439-447.

Chinnis AS, Williams JM, Treat KN. Electrical injuries. *In*: Tintinalli JE, Kelen GD, Stapczynski JS, eds. *Emergency Medicine: A Comprehensive Study Guide*. 5th ed. New York. McGraw-Hill, 2000,1292-1298.

Cunningham PA. The need for cardiac monitoring after electrical injury. *Med J Aust* 1991;134:765-766. Jain S, Bandi V. Electrical and lightning injuries. *Crit Care Clin* 1999;15:319-331.

Pimentel L, Mayo D. Lightning and electrical injuries. Critical Decisions in Emergency Medicine 2003;17:1-8.

RATIONALE (24)

Answer: A

Cellular phones placed in close proximity to an intensive care ventilator may be associated with malfunctions, including cessation of ventilator function. This is most likely to occur at very close distances (less than 30 cm) and during ringing. A recent study used a cell phone operated at 16, 40, 100, 250, 600 mW. Ventilator malfunctions occurring when cell phone was placed in very close proximity to the ventilator included an increase in the ventilatory rate, actual tidal volume increases, and an increase in the tidal volume display. In addition, the false activation of alarms was noted. There was only one case of a ventilator shutting down, which was noticed when the cellular phone was at maximum power output and less than 30 cm from the ventilator.

#### **REFERENCES (24)**

Irnich WE, Tobisch R. Mobile phones in hospitals. *Biomed Instrum Technol* 1999;33:28-34. Morrissey JJ, Swicord M, Balzano O. Characterization of electromagnetic interference of mobile devices in the hospital due to cell phones. *Health Phys* 2002;82:45-51.

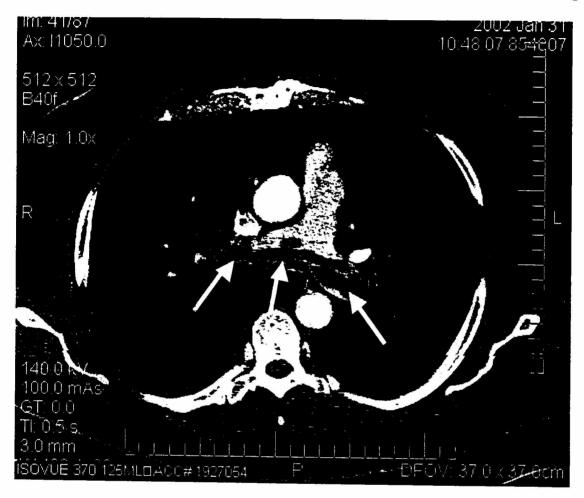
Shaw CI, Kacmarek RM, Hampton RL, et al. Cellular phone interference with the operation of mechanical ventilators. *Crit Care Med* 2004;32:928-931.

Turcotte J, Witters D. A practical technique for assessing electromagnetic interference in the clinical setting: ad hoc testing. *Biomed Instrum Technol* 1998;32:241-252.

RATIONALE (25)

Answer: B

This CT scan shows a large proximal (ie, "saddle") pulmonary embolism (see arrows in figure below).



The risk of mortality in massive pulmonary embolism (MPE) is directly related to the risk of a hemodynamic collapse from an acute right heart failure. Although the degree of hypoxemia (as estimated by Pao<sub>2</sub>:Fio<sub>2</sub> or A-a o<sub>2</sub> gradient) can be severe, hypoxic respiratory failure is rarely the cause of death. The pathophysiology of shock in MPE has been described in detail elsewhere, and can be briefly summarized as follows.

MPE increases the right ventricular (RV) outflow impedance. Because the RV cannot acutely generate mean pulmonary artery pressures greater than 40 mm Hg, the RV systolic function is impaired. This decreases left-sided filling pressures (left ventricular preload) and diminishes cardiac output. Shock occurs when catecholamine-induced vasoconstriction cannot maintain the mean arterial pressure.

The presence of hypotension would indicate a hemodynamically-significant PE. Not only would this indicate a higher mortality rate, but there is also a consensus that hypotension is the main indication for thrombolytic therapy. Although the decision to use thrombolytics based on ECG evidence is controversial in a normotensive patient, an ECG can provide valuable information. The presence of a right ventricular dysplasia (RVD) would indicate a hemodynamically-significant thromboembolism,

and would confer a much higher acute mortality risk. This information could help you risk-stratify a patient in terms of mortality. The absence of RVD would indicate a negligible acute mortality risk (unless there are recurrent thromboembolic events).

Patients with prior cardiopulmonary disease manifest a greater degree of hemodynamic sequelae for a relatively smaller clot burden. However, in patients without a history of prior cardiopulmonary disease, there is a consistent relationship between the degree of embolic obstruction and the propensity to develop shock. A prior history of cardiopulmonary disease would therefore confer a greater mortality risk for a significant thromboembolic event of any size.

#### REFERENCES (25)

- Hyers TM, Agnelli G, Hull RD, et al. ACCP Consensus Committee on Pulmonary Embolism: antithrombotic therapy for venous thromboembolic disease. *Chest* 2001;119:176S-193S.
- Goldhaber SZ, Haire WD, Feldstein, ML, et al. Alteplase versus heparin in acute pulmonary embolism: randomised trial assessing right-ventricular function and pulmonary perfusion. *Lancet* 1993;341:507-511.
- Goldhaber SZ. Pulmonary embolism. N Engl J Med 1998;339:93-104.
- Grifoni S, Olivotto I, Cecchini P, et al. Short-term clinical outcome of patients with acute pulmonary embolism, normal blood pressure, and echocardiographic right ventricular dysfunction. *Circulation* 2000;101:2817-2822
- Konstantinides S, Geibel A, Heusel G, et al. Heparin plus alteplase compared with heparin alone in patients with submassive pulmonary embolism. *N Engl J Med* 2002;347:1143-1150.
- Kreit JW. The impact of right ventricular dysfunction on the prognosis and therapy of normotensive patients with pulmonary embolism. *Chest* 2004;125:1539-1545.
- Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. *Chest* 2002;121:877-905.

## Appendices

### Appendix 1: Normal Laboratory Values

Laboratory Test	Specimen	Conventional Units	SI Units*
			···
I. Plasma, Serum, Whole Blood			
Albumin	serum	3.1-4.1 g/dL	31-41 g/L
Alkaline phosphatase (Alk Phos)	serum	30-85 IU/mL	0.75-1.92 μkat/L
Aminotransferase, alanine (ALT, SGPT)	serum	10-40 U/mL	0.17-0.67 μkat/L
Aminotransferase, aspartate (AST, SGOT)	serum	10-40 U/mL	0.17-0.67 μkat/L
Amylase Arterial studies (on room air):	serum whole blood	80-180 (Somogyi) U/dL	0.88-2.05 nkat/L
- pH		7.38-7.44	7.38-7.44
- PaCO <sub>2</sub>		35-45 mm Hg	4.7-6.0 kPa
- PaO <sub>2</sub>		75-100 mm Hg	10.0-13.3 kPa
- oxyhemoglobin saturation:		96%-100%	0.96-1.00
SaO <sub>2</sub>	co-oximeter		
SpO <sub>2</sub>	pulse oximeter		
Bicarbonate (HCO <sub>3</sub> )	serum	23-28 mEq/L	23-28 mmol/L
Bilirubin, direct	serum	0.0- $0.4$ mg/dL	0.0-7 μmol/L
Bilirubin, total	serum	0.0- $1.0$ mg/dL	0.0-17 μmol/L
Calcium (Ca)	serum	8.5-10.5 mg/dL	2.1-2.6 mmol/L
Calcium, ionized	plasma	1.14-1.30 mmol/L	1.14-1.30 mmol/L
Chloride (Cl)	serum	98-106 mEq/L	98-106 mmol/L
Creatine kinase (CK) -total	serum	5-55 U/L	0.67-2.50 μkat/L
-MB isoenzymes		<5% total	<5% total
Creatinine (Cr)	serum	0.7-1.5 mg/dL	53-133 μmol/L
Digoxin (therapeutic range)	serum	0.9-2.0 ng/mL	0.9-2.0 ng/mL
Fibrinogen	plasma	200-400 mg/dL	o., z.o ng mb
Glucose, fasting	plasma	70-110 mg/dL	3.9-6.1 mmol/L
Hematocrit (Hct)	whole blood	37%-52%	0.37-0.52
Hemoglobin (Hgb)	whole blood	12-18 g/dL	7.4-11.2 mmol/L
Lactate, venus	plasma	0.5-2.2 mEq/L	0.5-2.2 mmol/L
Lactate dehydrogenase (LDH)	serum	60-100 U/mL Wacker U	1.83-3.50 µkat/L

Laboratory Test	Specimen	Conventional Units	SI Units*
Leukocyte count (WBC)	whole blood	4.3-10.8	4.3-10.8
-neutrophils		50%-70%	0.50-0.70
-bands		0%-5%	0.0-0.05
-eosinophils		0%-3%	0.0-0.03
-basophils		0%-1%	0.0-0.01
-lymphocytes		30%-45%	0.30-0.45
Osmolality	serum	280-296 mOsm/kg H <sub>2</sub> O	280-296 mmol/kg
Partial thromboplastin time, activated (aPTT)	plasma	24-37 secs	24-37 secs
Phosphorus	serum	2.6-4.5 mg/dL	0.84-1.45 mmol/L
Platelet count	whole blood	150-350 x 10 <sup>3</sup> /mm <sup>3</sup>	150-350 x 10 <sup>9</sup> /L
Potassium (K)	serum	3.5-5.0 mEq/L	3.5-5.0 mmol/L
Proteins	serum	-	
-albumin		3.1-4.3 g/dL	31-43 g/L
-total		6.0-8.0 g/dL	60-80 g/L
Prothrombin time (PT)	plasma	8.8-11.6 secs	8.8-11.6 secs
Sodium (Na)	serum	135-145 mEq/L	135-145 mmol/L
Urea nitrogen, blood (BUN)	serum	8-25 mg/dL	2.9-8.9 mmol/L
II. Urine			
Osmolality	urine	38-1,400 mOsm/kg H <sub>2</sub> O	38-1,400 mOsm/ kg H,O
Sodium	urine	varies with intake	varies with intake

# Appendix 2: Abbreviations, Acronyms, Symbols, and Units

ABG arterial blood gas

AC assist-control (volume) ventilation
ACLS advanced cardiac life support
ACTH adrenocorticotropin hormone

ADH antidiuretic hormone

AIDS acquired immunodeficiency syndrome antineutrophilic cytoplasmic antibodies

AP anteroposterior

aPTT activated partial thromboplastin time (sec)
ARDS acute (adult) respiratory distress syndrome

ARF acute renal failure
ATN acute tubular necrosis
BAL bronchoalveolar lavage

BiPAP biphasic positive airway pressure bone marrow transplantation

BP systemic (arterial) blood pressure (mm Hg)

BSA body surface area (m²) °C degrees Celsius

Cao<sub>2</sub> arterial content of Oxygen (mL O<sub>2</sub>/dL)

CCU coronary care unit
cfu colony forming units
CHF congestive heart failure
CHI closed head injury
CI cardiac index (L/min/m²)

cm centimeter
CMV cytomegalovirus
CNS central nervous system
CO cardiac output (L/min)

CO<sub>2</sub> carbon dioxide

COPD chronic obstructive pulmonary disease

CPAP continuous positive airway pressure (cm H<sub>2</sub>O)

CPR cardiopulmonary resuscitation

CSF cerebrospinal fluid C-Spine cervical spine

CT computed tomography
CUS compression ultrasonography

C<sub>v</sub>O<sub>v</sub> venous content of oxygen (mL O2/dL

DI diabetes insipidus

DIC disseminated intravascular coagulation

DKA diabetic ketoacidosis

dL deciliter

DNR do-not resuscitate

DO<sub>2</sub> oxygen delivery (mL O<sub>2</sub>/min)
DPL diagnostic peritoneal lavage
DVT deep venous thrombosis

ECMO extracorporeal membrane oxygenation

EEG electroencephalogram EKG electrocardiogram

EPAP expiratory positive airway pressure

ERCP endoscopic retrograde cholangiopancreatography

ETOH ethyl alcohol (ethanol)
°F degrees Fahrenheit

FEV forced expiratory volume in one second (mL)

FFP fresh frozen plasma

Fig. fractional inspired concentration of oxygen

FVC forced vital capacity (mL)

GI gastrointestinal
GCS Glasgow Coma Scale

Hct hematocrit (%)

HEENT head, eyes, ears, nose, throat

HELLP hemolysis, elevated liver enzymes, low platelets

Hgb hemoglobin (g/dL)

HIV human immunodeficiency virus

hpf high-power field

H<sub>2</sub>O water

HSP Henoch-Schonlein purpura
HR heart rate (beats/min)

HUS hemolytic uremic syndrome

ICP intracranial pressure ICU intensive care unit

I:E inspiratory time-expiratory time ratio IMV intermittent mandatory ventilation

INH isoniazid

INR international normalized ratio
IPAP inspiratory positive airway pressure

IPG impedance plethysmography

IPP inspiratory plateau pressure (cm H<sub>2</sub>O)

IRV inverse ratio ventilation

IV intravenous

IVC inferior vena cava

kg kilogram L liter

LDH lactic dehydrogenase (U/mL)

LP lumbar puncture LV left ventricle

m meter

MAP mean arterial pressure (mm Hg)
MAT multifocal atrial tachycardia

milligram mg MG myasthenia gravis malignant hyperthermia MH MI myocardial infarction mLmilliliter mm millimeter  $mm^3$ cubic millimeter multiple organ dysfunction syndrome **MODS** MRI magnetic resonance imaging microgram μg NG nasogastric **NMBA** neuromuscular blocking agents **NPPV** noninvasive positive-pressure ventilation ng nanogram 0, oxygen ΟÍ oxygenation index P heart rate (beats/min) partial pressure of oxygen of arterial blood (mm Hg) Pao, partial pressure of carbon dioxide of arterial blood (mm Hg) Paco, PA posterioanterior pulmonary artery hypertension PAH PAN polyartertis nodosa pulmonary artery occlusion (wedge) pressure (mm Hg) **PAOP** PAP pulmonary artery pressure (mm Hg) **PCA** patient-controlled analgesia **PCP** Pneumocystic carinii pneumonia **PCV** pressure-controlled ventilation PE pulmonary embolism **PEEP** positive end-expiratory pressure (cm H<sub>2</sub>O) end-tidal carbon dioxide tension (mm Hg) Petco. **PICU** pediatric intensive care unit PIP peak inflationary pressure (cm H<sub>2</sub>O) pRBC packed red blood cells **PSB** protected specimen brush **PSV** pressure-support ventilation PT prothrombin time PTT partial thromboplastin time partial pressure of oxygen of mixed-venous blood (mm Hg) Pvo. Q blood flow (L/min) RA rheumatoid arthritis **RAP** right atrial pressure (mm Hg) **RBC** red blood cell REE resting energy expenditure RQ respiratory quotient RR respiratory rate/minute RV right ventricle oxyhemoglobin saturation of arterial blood (%) (measured by co-oximeter) Sao, SAH subarachnoid hemorrhage

SG specific gravity

SIMV synchronized intermittent mandatory ventilation SIRS systemic inflammatory response syndrome

Spo, oxyhemoglobin saturation (%) (measured by pulse oximeter)

stat statim (immediately)

Svo, oxyhemoglobin saturation of mixed venous blood (%)

SVR systemic vascular resistance (dynes·sec/cm<sup>5</sup>)

T temperature TB tuberculosis

TCA tricyclic antidepressant
TIA transient ischemic attack
TMJ temporomandibular joint
TPA tissue plasminogen activator
TPN total parenteral nutrition

TTP thrombotic thrombocytopenic purpura

UA urinalysis

VC vital capacity (mL)

VE minute ventilation (L/min)

V<sub>D</sub> dead space (mL)

Vd volume of distribution (L) VD/VT dead space/tidal volume

Vo, oxygen consumption (mL O<sub>2</sub>/min)

V/Q ventilation/perfusion
Vt tidal volume (mL)
WBC white blood cell